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April 1946

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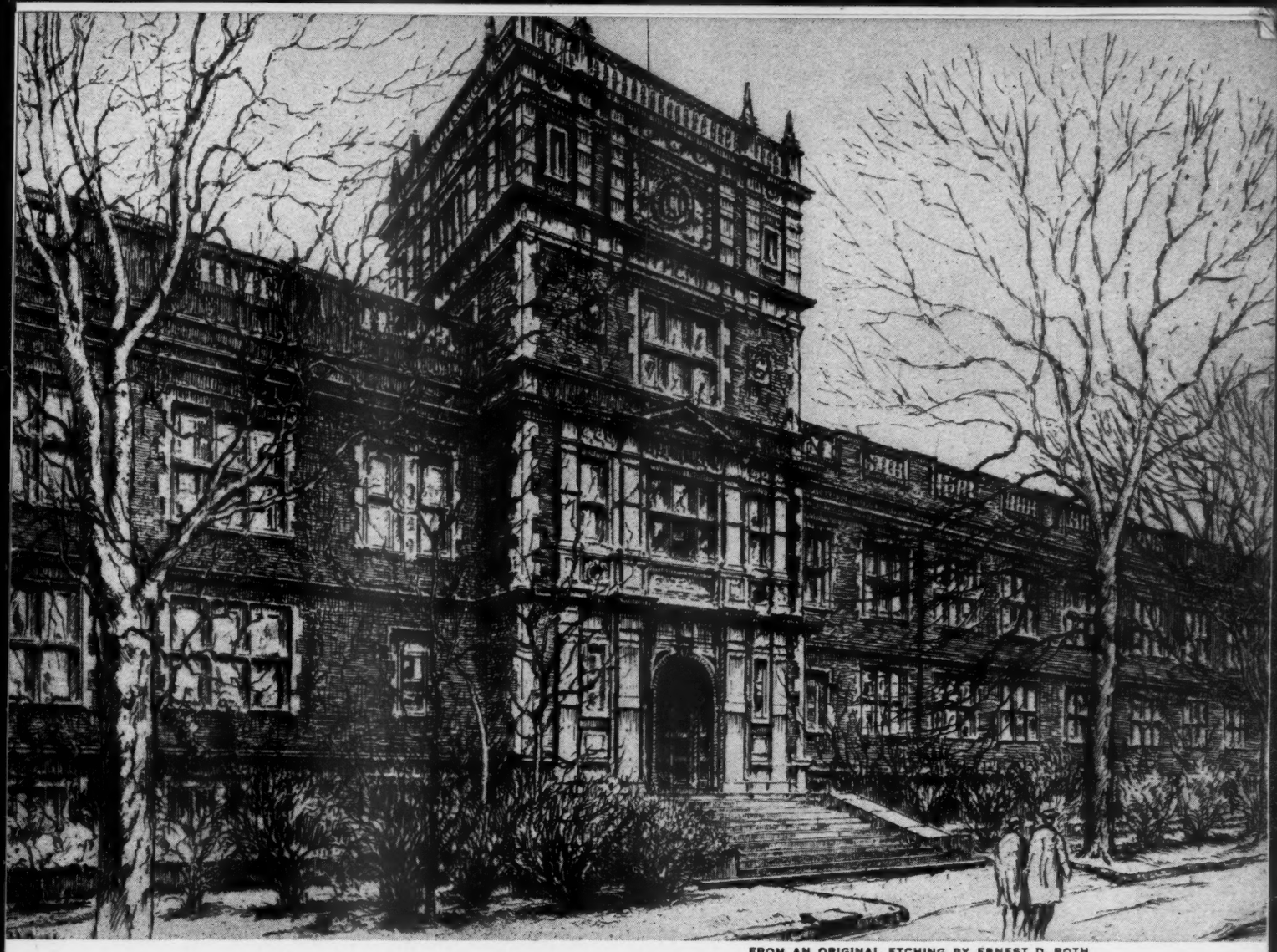

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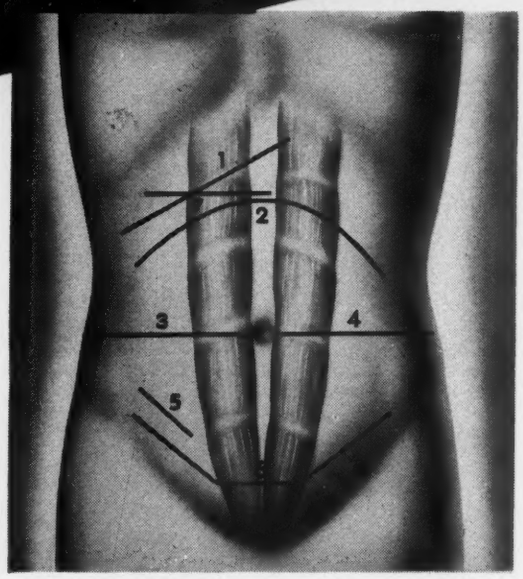

1. For gall bladder, common duct, head of pancreas.
2. Resection of stomach, transverse colon.
3. Resection of ascending colon, cecum, right sided lesions.
4. Descending colon, spleen and left sided lesions.
5. Appendectomy.
6. Pelvic surgery, resection of sigmoid.
7. Small exploratory transverse incision.

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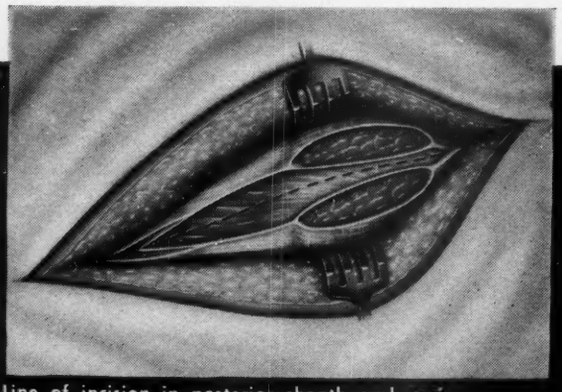
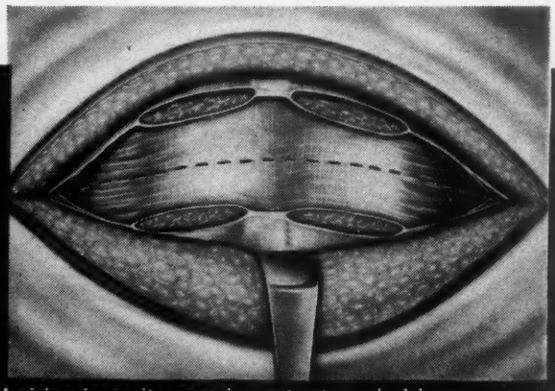
A resumé of the principles involved, together with some pictures describing the incisions, are shown on these two pages. This series covers the principles as they relate to the abdominal section of the body.



Line of incision in posterior sheath and peritoneum in gall bladder incision.



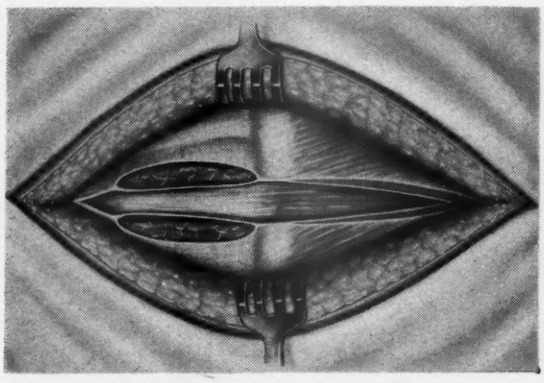
Incision in peritoneum in gastrectomy incision.

ADVANTAGES OF TRANSVERSE INCISIONS

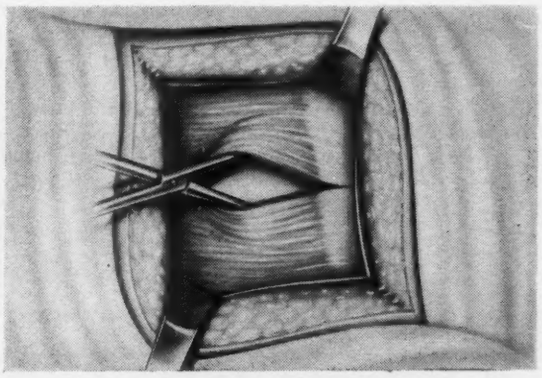
SUTURES hold more securely if the connective tissue layers within the wound are approximated in such a manner that the sutures must pull against the fibers within them. Sutures pull against the fibers only when the connective tissues are cut parallel to the direction of the fibers. In the anterior abdominal wall, this is usually best accomplished through a transverse incision. As a result, sutured transverse incisions are stronger than sutured vertical incisions.

In the past, transverse abdominal incisions did not find a wide acceptance because they necessitated cutting across muscle. Attempts were made to suture the muscles and the sutures not only held poorly, but they almost invariably caused necrosis.

In the new technic, no attempt is made to suture muscles—only the surrounding sheath. Necrosis is thereby avoided. Some approximation of muscle is obtained by suturing their sheath, and further approximation is accomplished by keeping the patient in a semi-reclining position with knees partially reflexed.

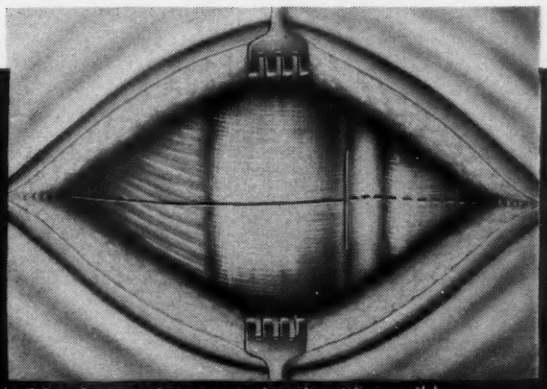


Cut rectus muscle in the incision for resecting the descending colon.

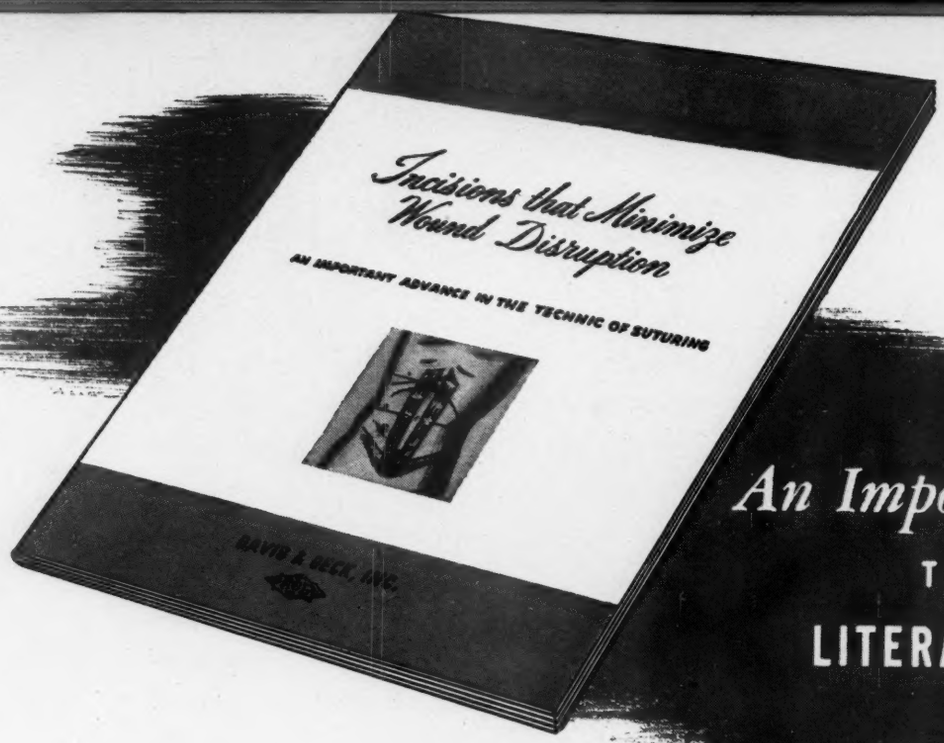


Separating muscle fibers in McBurney incision.

Cutting the fascia of the anterior sheath in lower abdominal transverse incision.



Incision in anterior rectus sheath with possible extension.



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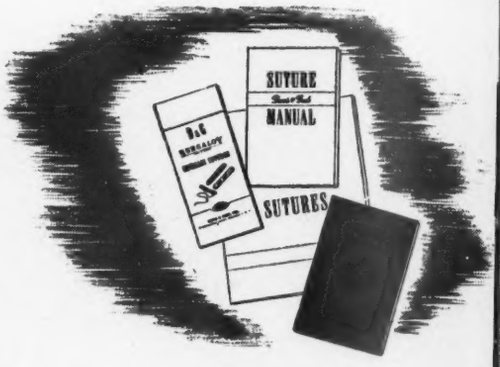

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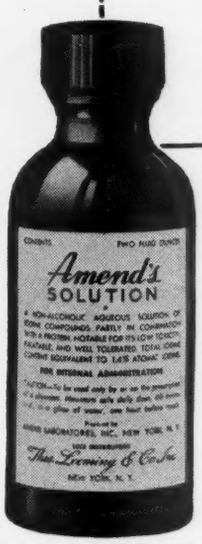

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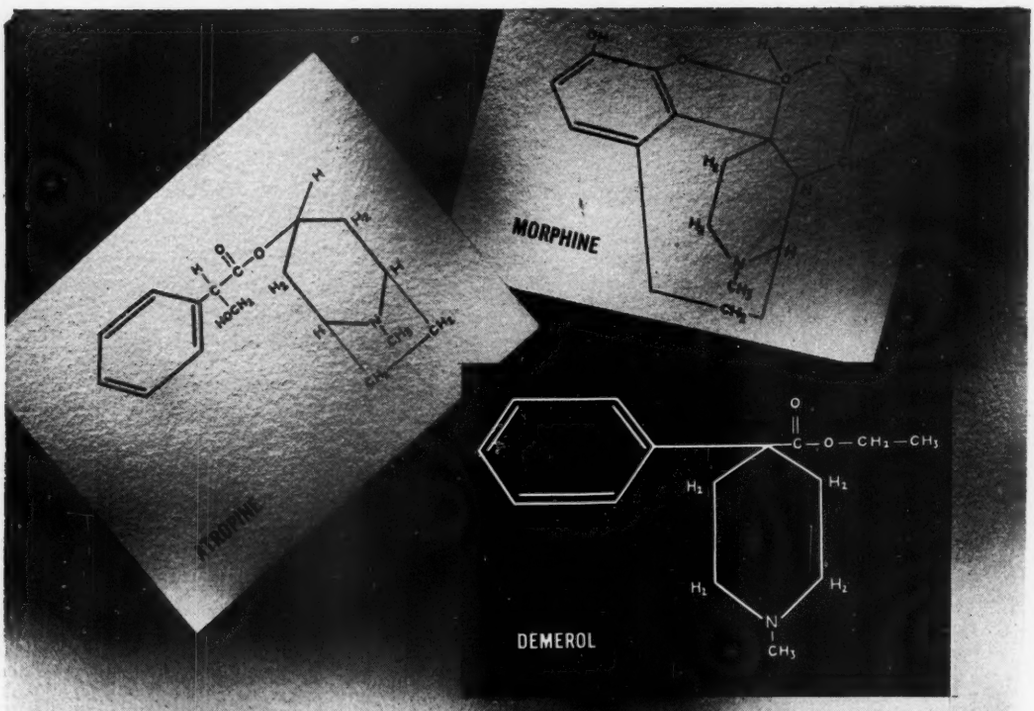

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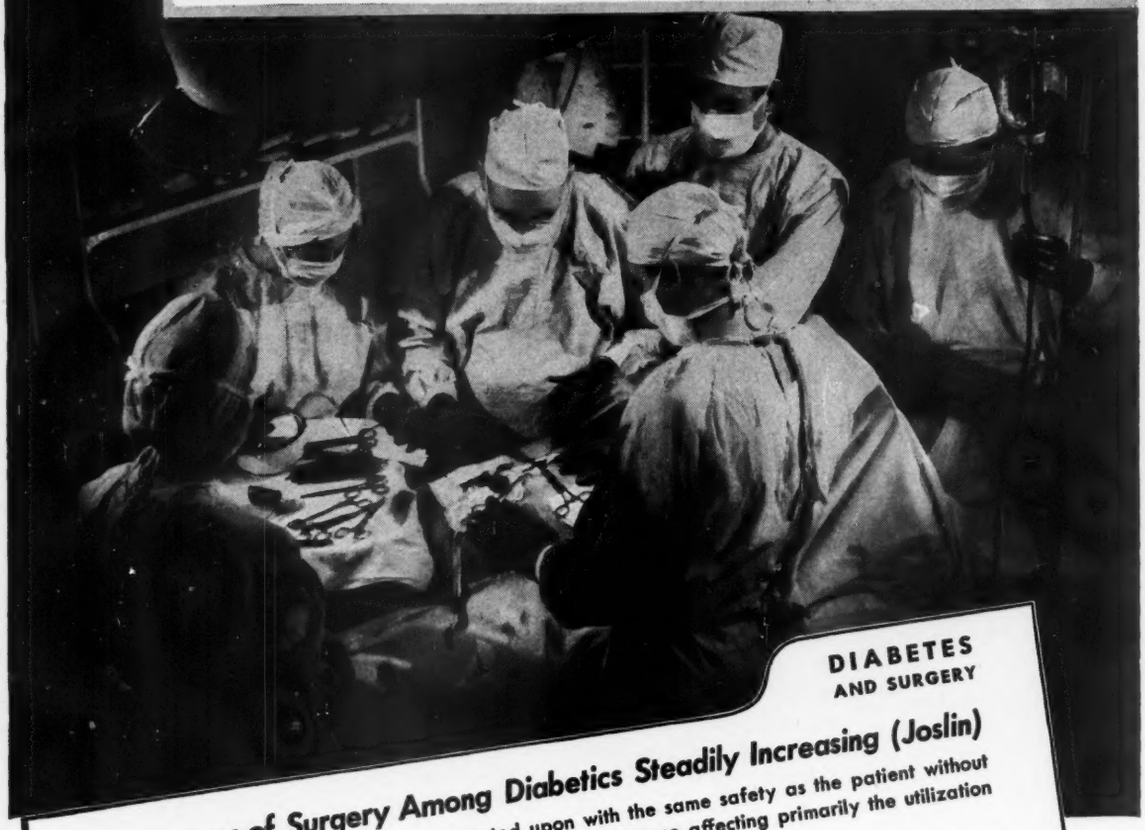

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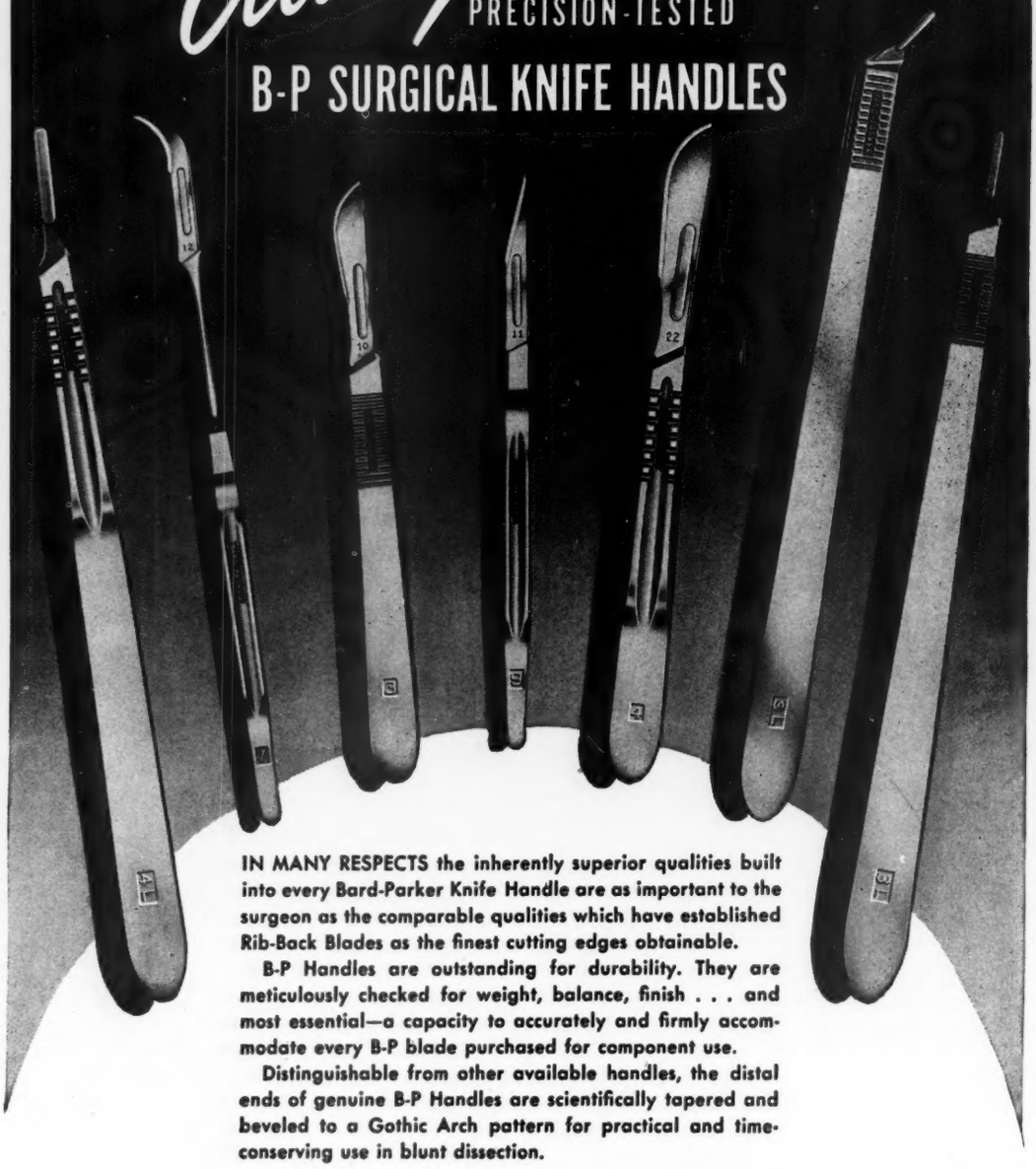

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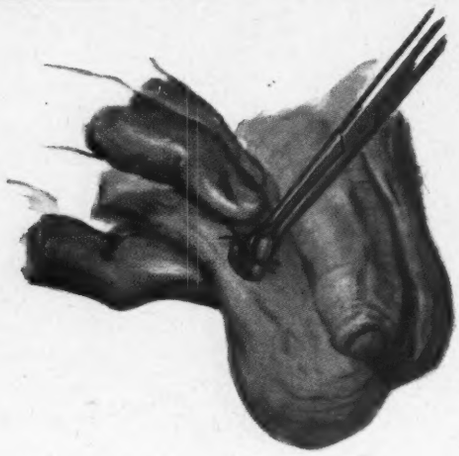
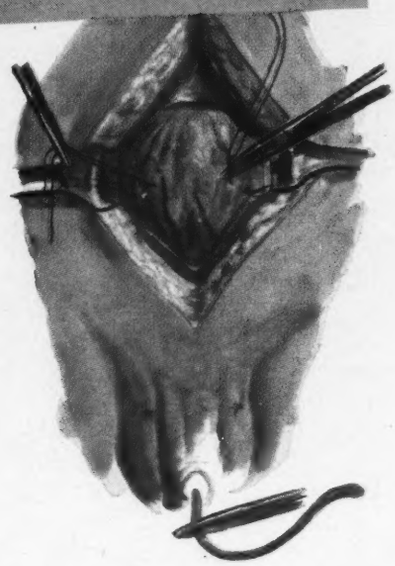
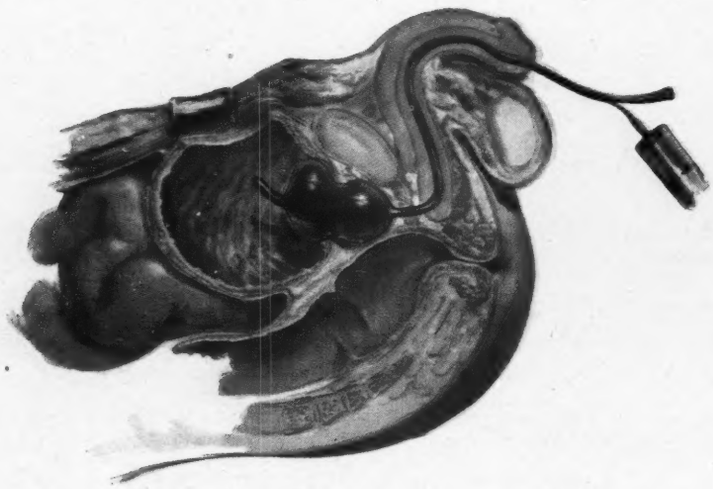


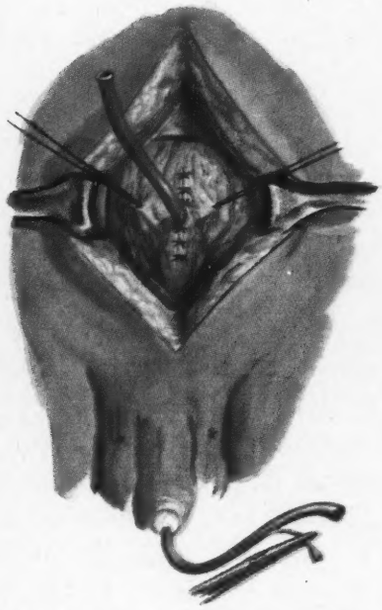
I. Bilateral vas-ligation and section performed.



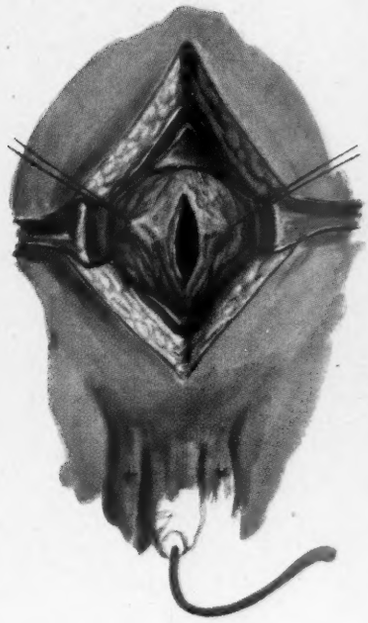
II. Bladder distended with water per catheter . . . exposed by midline suprapubic incision. Peritoneum stripped away upwards and laterally. Traction sutures placed.



V. Hemostatic bag-catheter distended in prostatic bed to control bleeding.

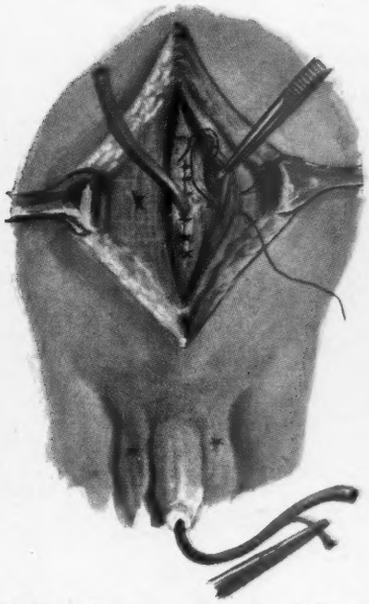


VI. Bladder closed around suprapubic tube, permitting later through and through irrigation.

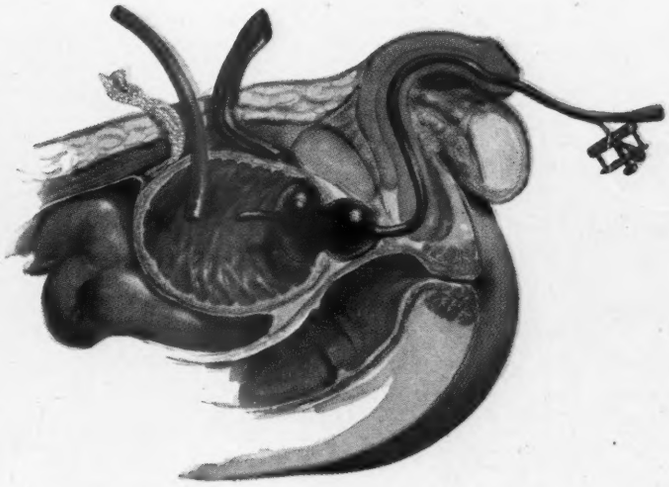


III. Bladder emptied per catheter . . . opened by vertical incision.

IV. Prostate shelled out digitally with counter pressure through rectum.



VII. Bladder fixed to anterior abdominal wall using previously placed traction sutures tied over rectus sheath.



VIII. Schema of completed operation showing suprapubic and sub-peritoneal drainage.

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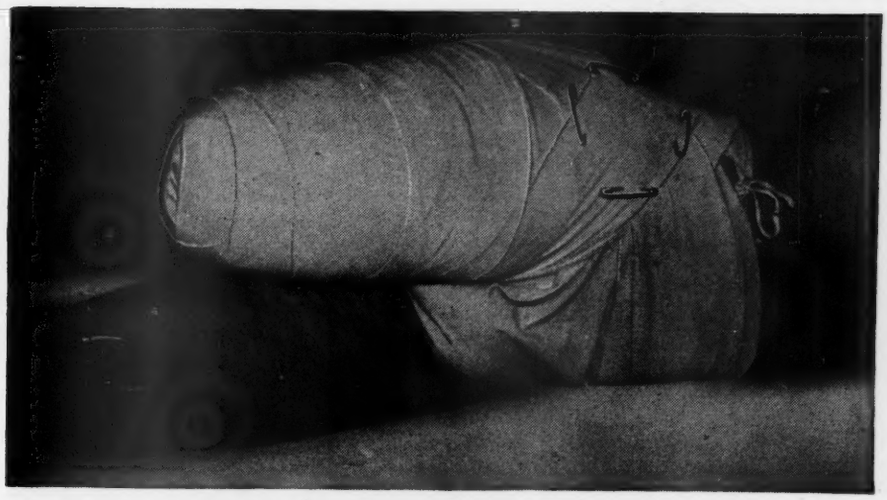

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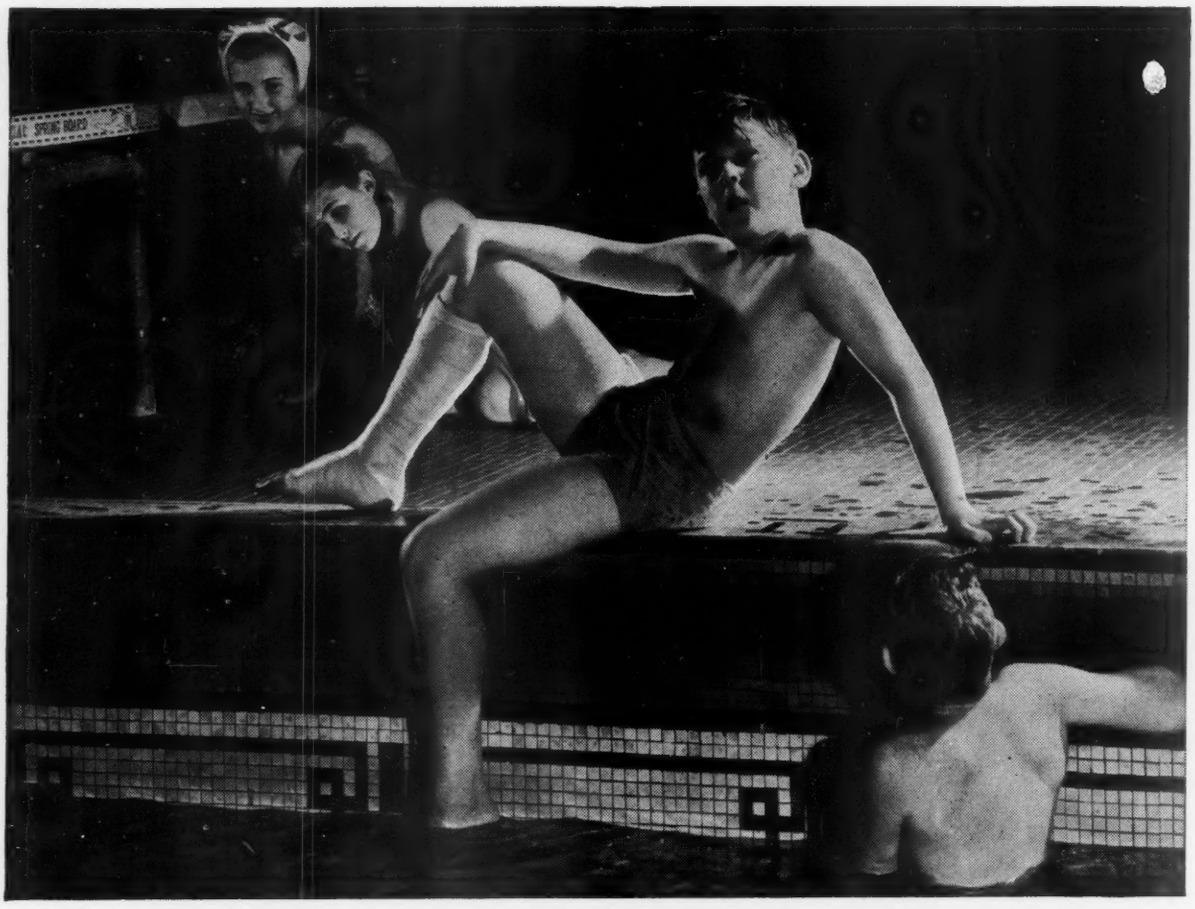

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He was a brave youngster. Fighting back tears, he asked whether he'd have to stay home while the others had all the fun.

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Fiberglas yarn in the bandage of which the cast is made, prevents the cast from changing size as the setting solution dries. And because the fine fibers of glass are not affected by moisture and are extremely strong, these open-mesh casts are light, cool and can be worn while bathing.*

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* Known as "Aire-Lite", the Fiberglas-plastic bandage for waterproof casts is described by Roger Anderson, M.D., and Herbert R. Erickson, M.S. in *The American Journal of Surgery*, LXIX, 3 (September, 1945), pp. 299-305. Write us for a reprint.

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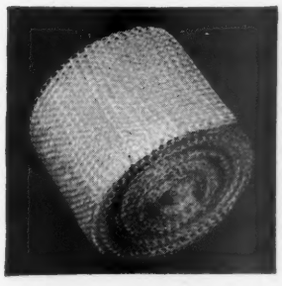

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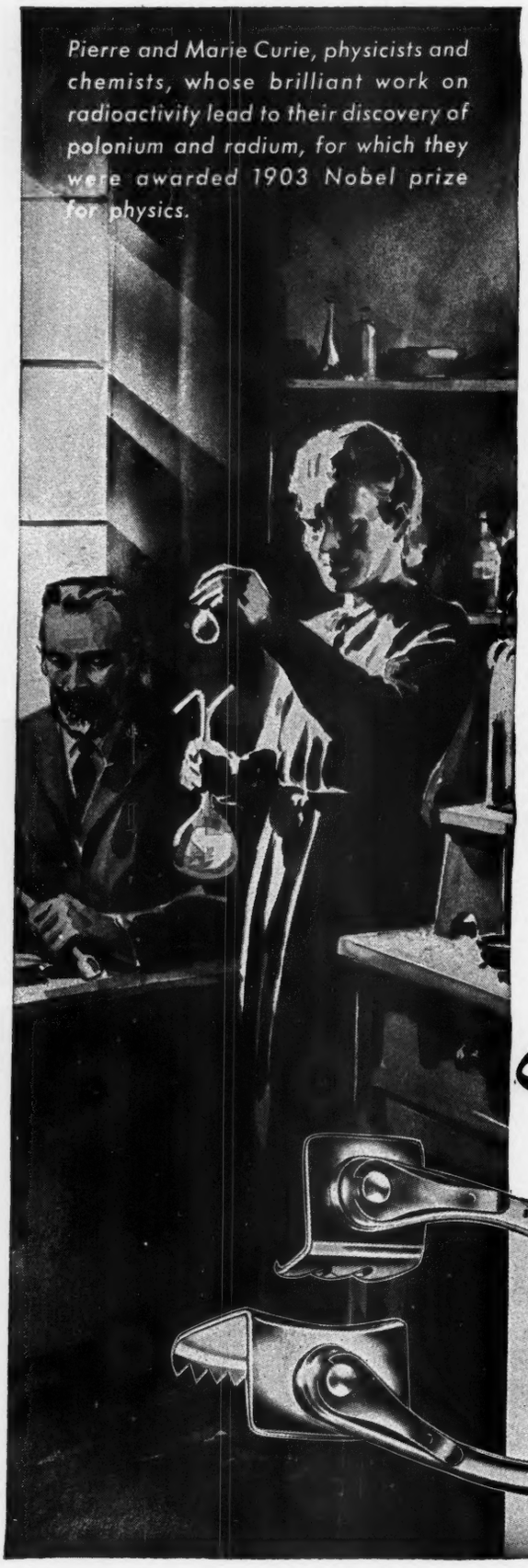

Pierre and Marie Curie, physicists and chemists, whose brilliant work on radioactivity lead to their discovery of polonium and radium, for which they were awarded 1903 Nobel prize for physics.

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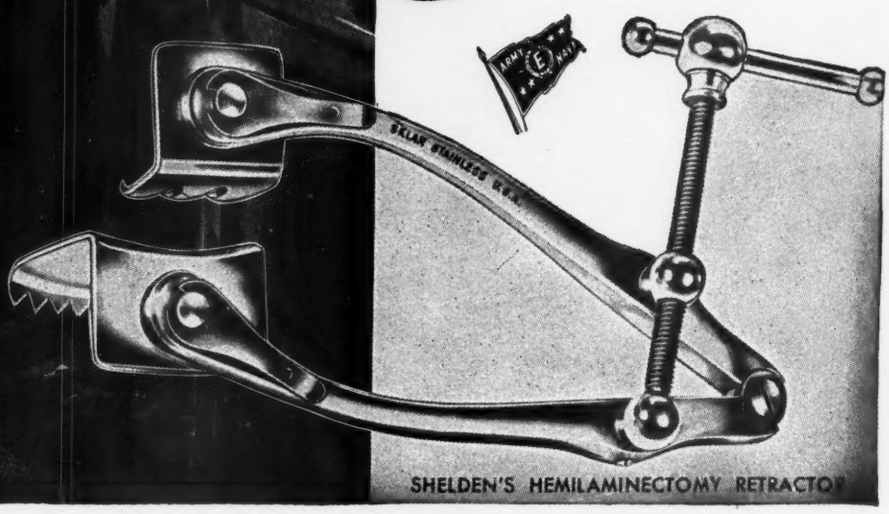

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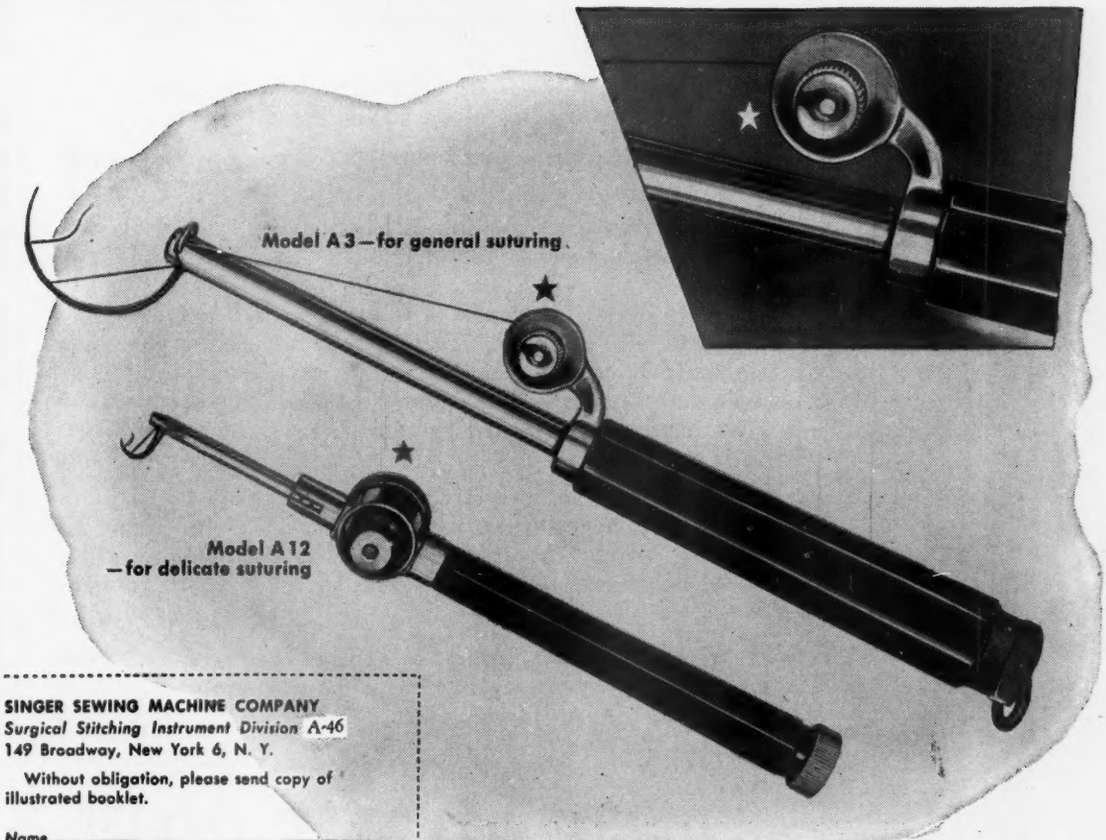

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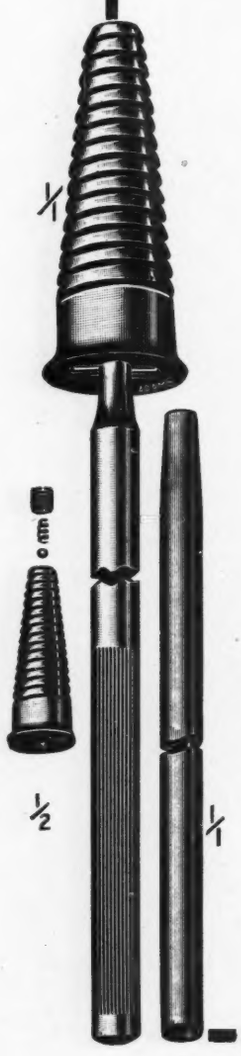

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—Mc GOVERN, F. H.: ARCH. OTOLARYNGOLOGY, *40*:196-197 (SEPT.) 1944

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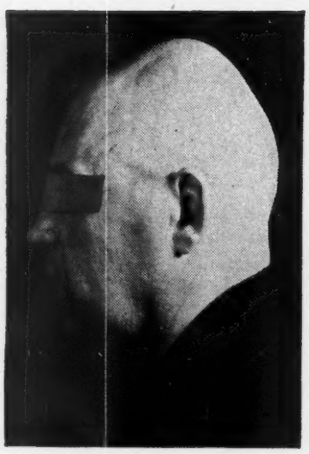
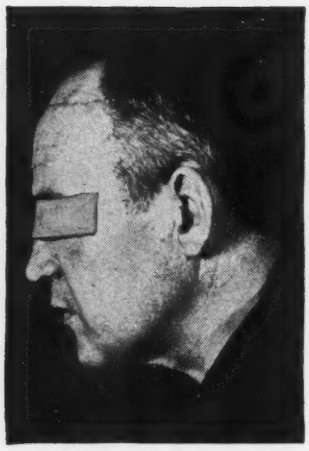


Lateral view of patient before operation for insertion of tantalum implant.



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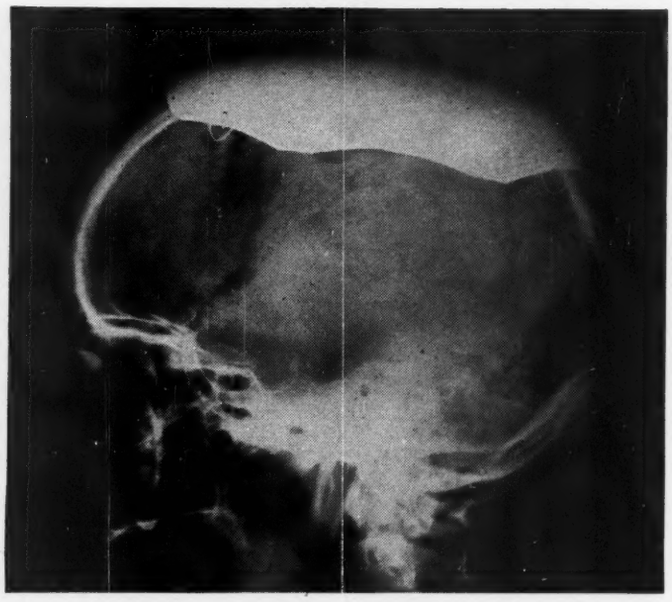
† Gardner, W. James, M. D., F. A. C. S., "Closure of Defects of the Skull with Tantalum," S. G. & O., 80: 303, March, 1945.



Postoperative roentgenogram of patient shown above.

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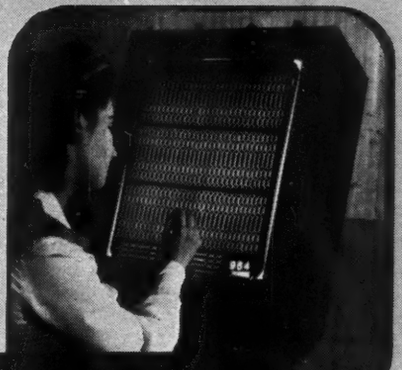

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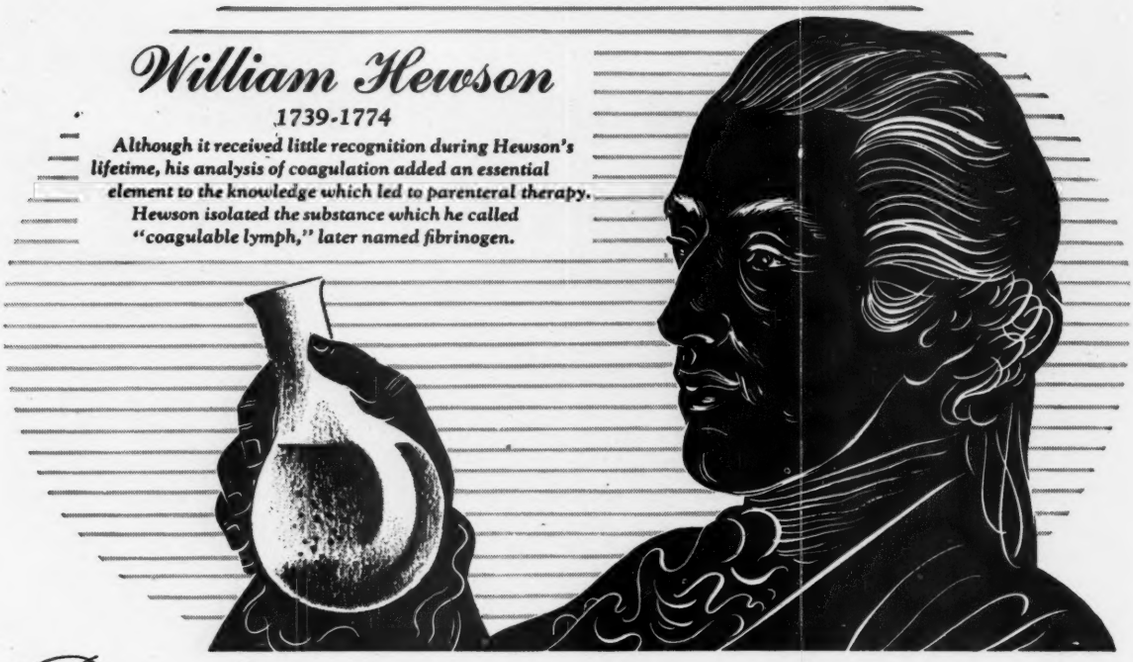

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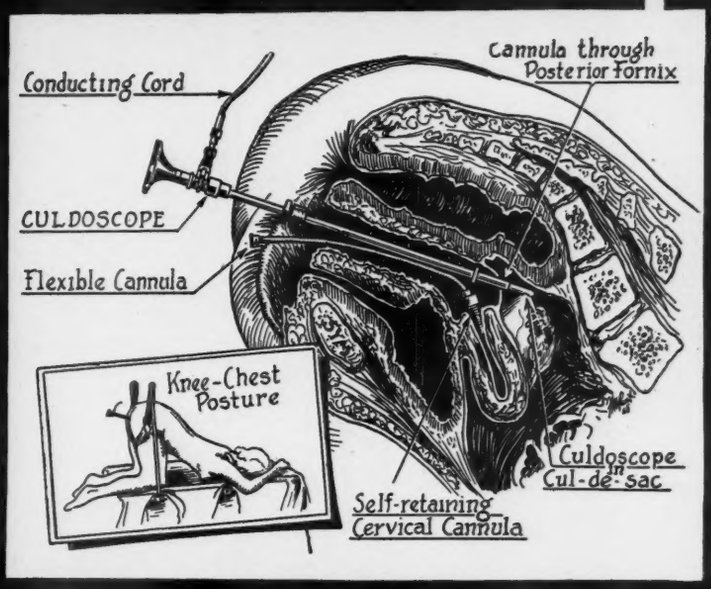

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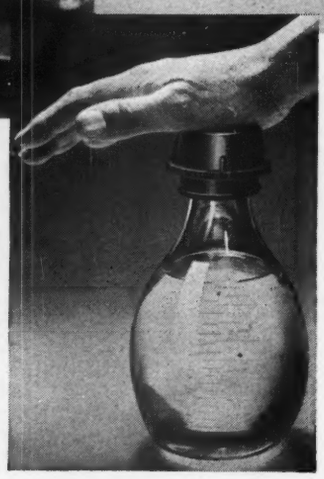

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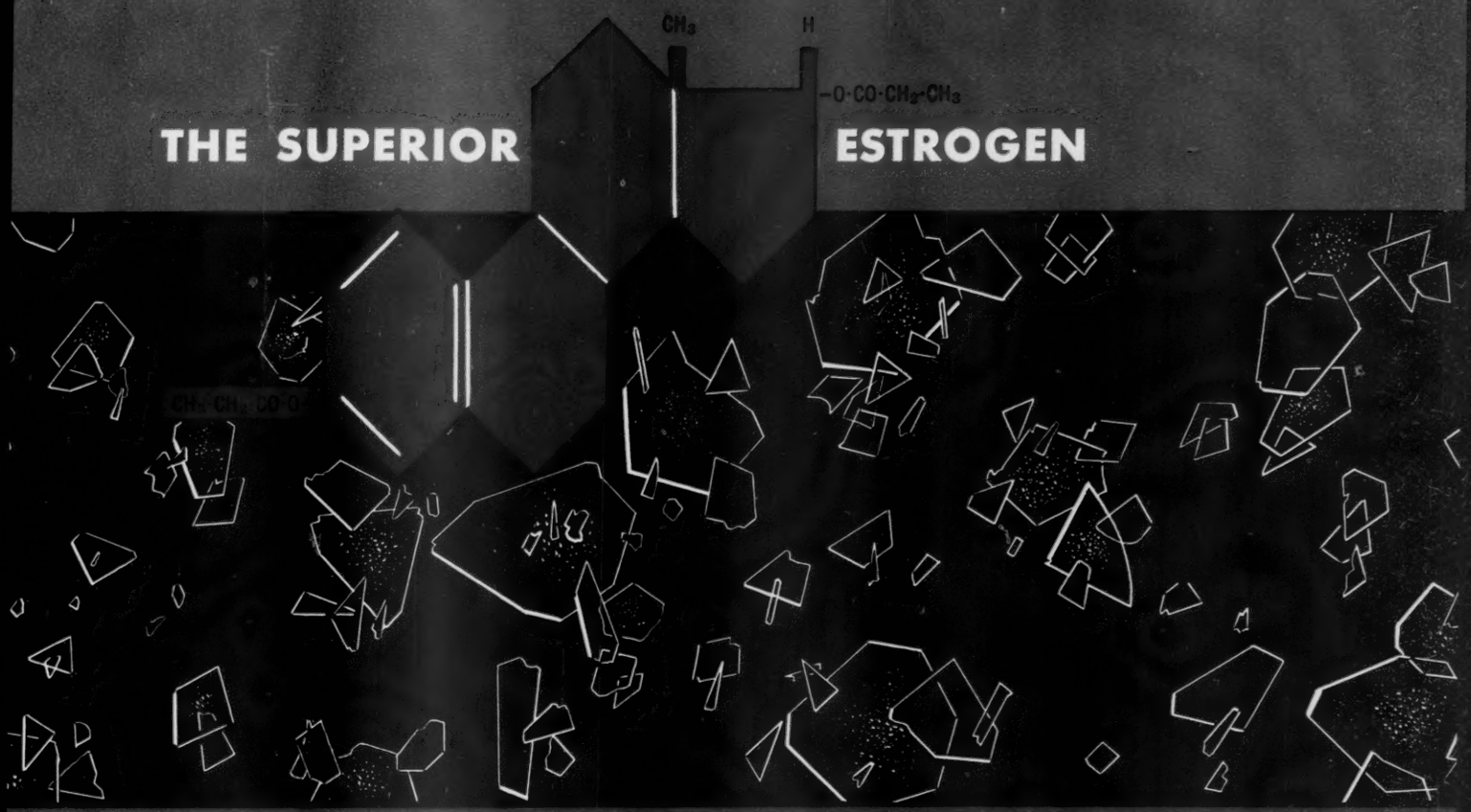

Ciba research is proud of its accomplishments in the field of steroid sex hormones. The development of endocrinology from the early days when de Graaf gave the first authentic account of the ovarian follicle, to the present-day understanding of the vital role played by the gonadal hormones, is an evolution in which Ciba has latterly had an important part. The record of Ruzicka and his associates, who first synthesized the androgenic hormone in cooperation with Ciba laboratories, is one of the cornerstones of our tradition.

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With the development of Di-Ovocylin, Ciba offers the superior estrogen having long-sought advantages of prolonged duration of effect with greatest economy.

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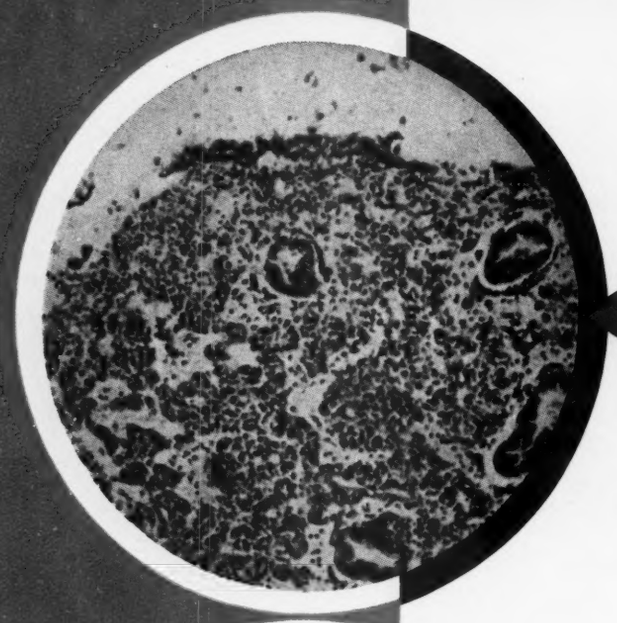
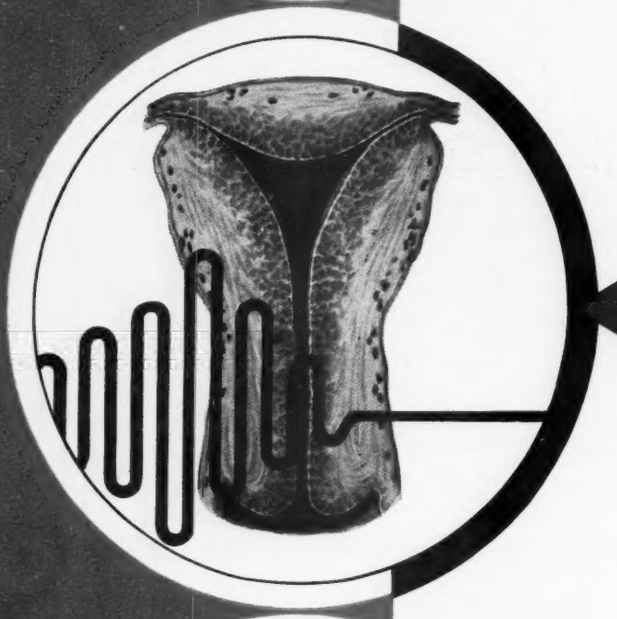


INFANTILISM:
ENDOMETRIAL
BIOPSY

AMENORRHEA

"While not establishing spontaneous menstrual periods," large doses of estrogenic substances "nevertheless resulted in the appearance of many episodes of uterine bleeding, breast development, development of pubic and axillary hair and physiological and social adjustments."

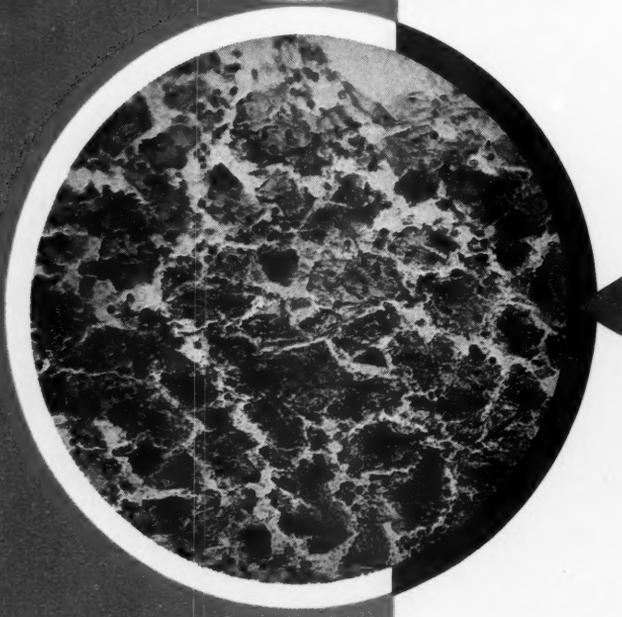
Finkler & Bass: Med. Woman's J., 47: 1, 1940.



DYSMENORRHEA

"The cramps of essential dysmenorrhea can be successfully eliminated with estrogen when an adequate dose is started early enough in the cycle. The advantages of using 'estradiol dipropionate' are that a minimum of injections are required and there are no demonstrable toxic effects."

Sturgis & Meigs: Surg., Gyn., & Obst., 75: 87, 1942.



MENOPAUSE:
VAGINAL SMEAR

MENOPAUSE

It is suggested that menopausal patients "should receive injections of 5 mg. of estradiol dipropionate" . . . "once weekly for three weeks. If this does not produce a satisfactory therapeutic response there is no need to continue searching for the proper dosage of estrogens, since this dose is ample for all such cases."

Queries and Minor Notes, J. Am. Med. Assoc., 129: 1295, Dec. 29, 1945.

DI-OVOCYLIN

GIVES INCREASED INTENSITY AND PROLONGED DURATION OF EFFECT

Early estrogen therapy was hampered by the short-lived effect of the injection. Increasing doses merely resulted in wastage. Small divided amounts meant inconvenient multiple injections. With the advent of the esterified estrogenic hormone, Di-Ovocylin (estradiol dipropionate), the organism is spared the periodic alternation of hormonal want or abundance, and is supplied a constant, sufficient amount of estrogen. Whereas estrone injections may be required two or three times weekly, a single injection of Di-Ovocylin every fourteen to twenty-one days will control symptoms in the majority of menopausal patients.[1] In potency as well as in duration of effect, Di-Ovocylin offers greatest advantage. In one comparison of the potency of several estrogens, using the oviduct-stimulating power in chicks as criterion, it was shown that estrone had the lowest potency, estradiol and estradiol benzoate were higher, and estradiol dipropionate had highest potency.[2] Make Di-Ovocylin your estrogen of choice for its lasting therapy, easier management of cases, and gratifying economy.

PRODUCES FEELING OF WELL-BEING

Di-Ovocylin, being a derivative of a natural estrogenic hormone, has the added advantage of producing a feeling of well-being in the patient. This important clinical consideration further recommends Di-Ovocylin for use as your regularly prescribed estrogen.

1. Greene, R. R.: Int. Abst. Surg., 74: 595, 1942.
2. Monro, S. S., Kosin, I. L.: Endocrinology, 27: 687, 1940.

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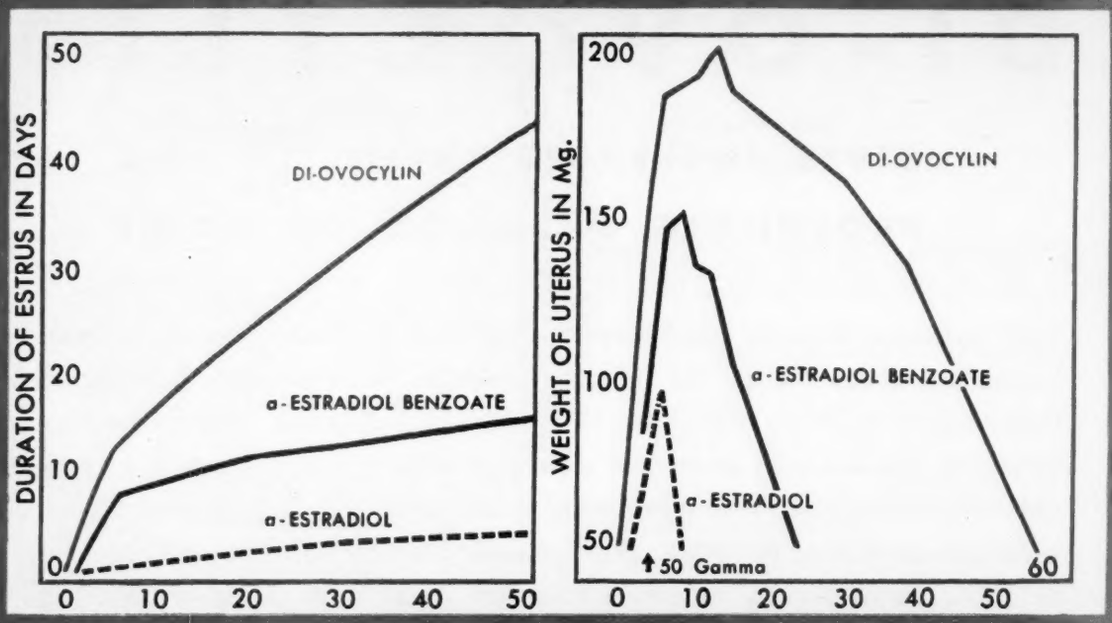

Duration of estrus produced in castrated rats by estradiol, estradiol benzoate, and estradiol dipropionate.

Effect of estradiol, estradiol benzoate, and estradiol dipropionate on the uterine weight of castrated rats.

CIBA ESTROGENS

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(α-estradiol dipropionate)

Supplied: Ampuls of 1 cc. containing 0.1 or 0.2 mg., in cartons of 6 and 50; ampuls of 1 cc. containing 0.5, 1.0, 2.5, or 5.0 mg., in cartons of 3, 6, and 50.

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OVOCYLIN SUPPOSITORIES

Used in conditions such as vulvovaginitis of infants, senile vaginitis, pruritus vulvae. Supplied: Suppositories of 0.04 mg., boxes of 10 (Adult's size) and 30 (Children's size); 0.4 mg., boxes of 10 (Adult's size).

OVOCYLIN OINTMENT

For concentrated local effect in conditions such as under-developed breasts or senile vaginitis. Supplied: Concentrations of 0.03 mg. and 0.15 mg. per Gram, in tubes of 50 Grams.

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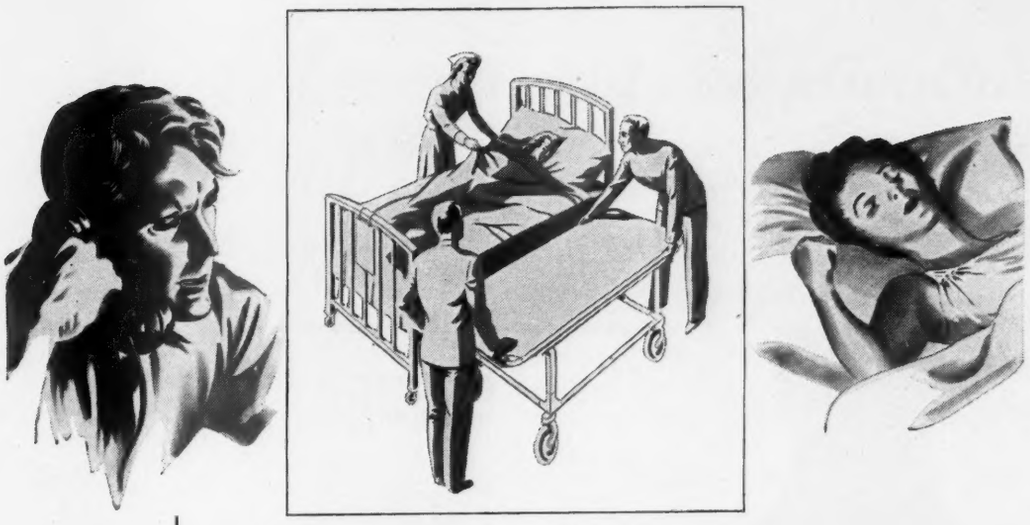

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Combisul-DM available in 0.5 Gm. tablets. Bottles of 100 and 1000.

1. Lehr, D.: Proc. Soc. Exper. Biol. & Med. **58**:11, 1945.
2. Lehr, D.: In press.

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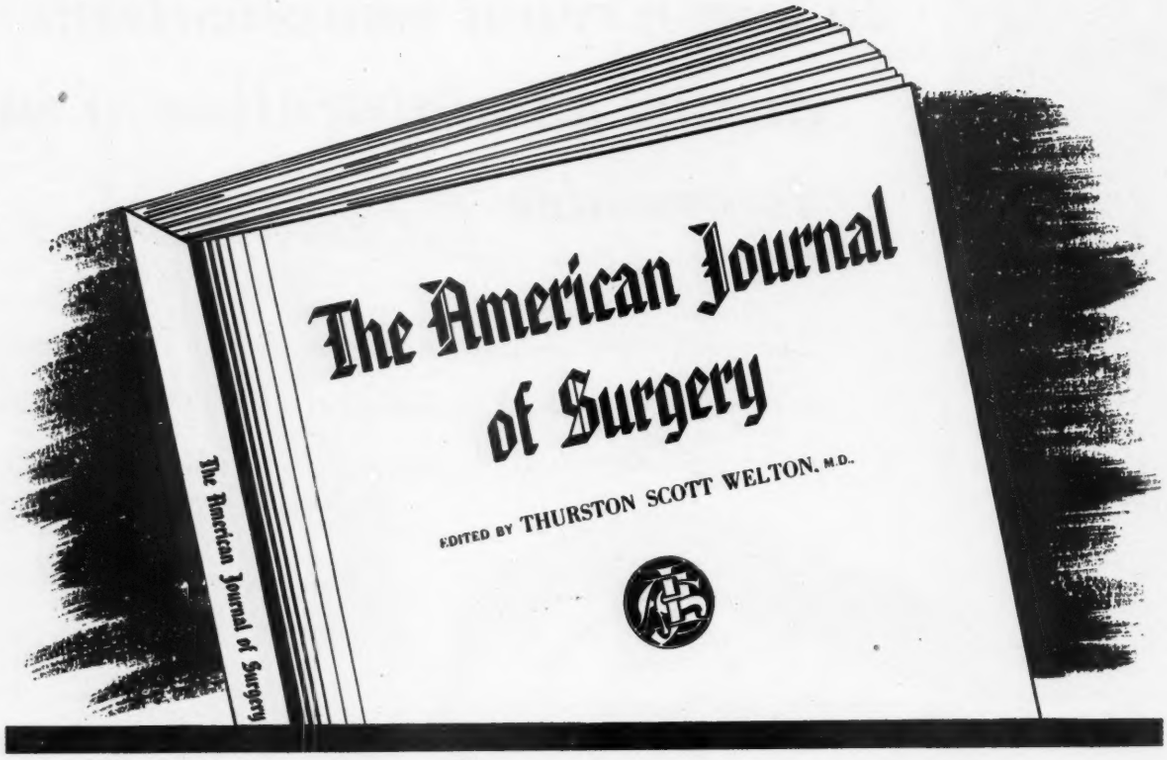

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**Kelly, M.: Anesth. & Analg. 19:107, 1940.*

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†Rochberg, S.: Anesth. & Analg. 22:174, 1943

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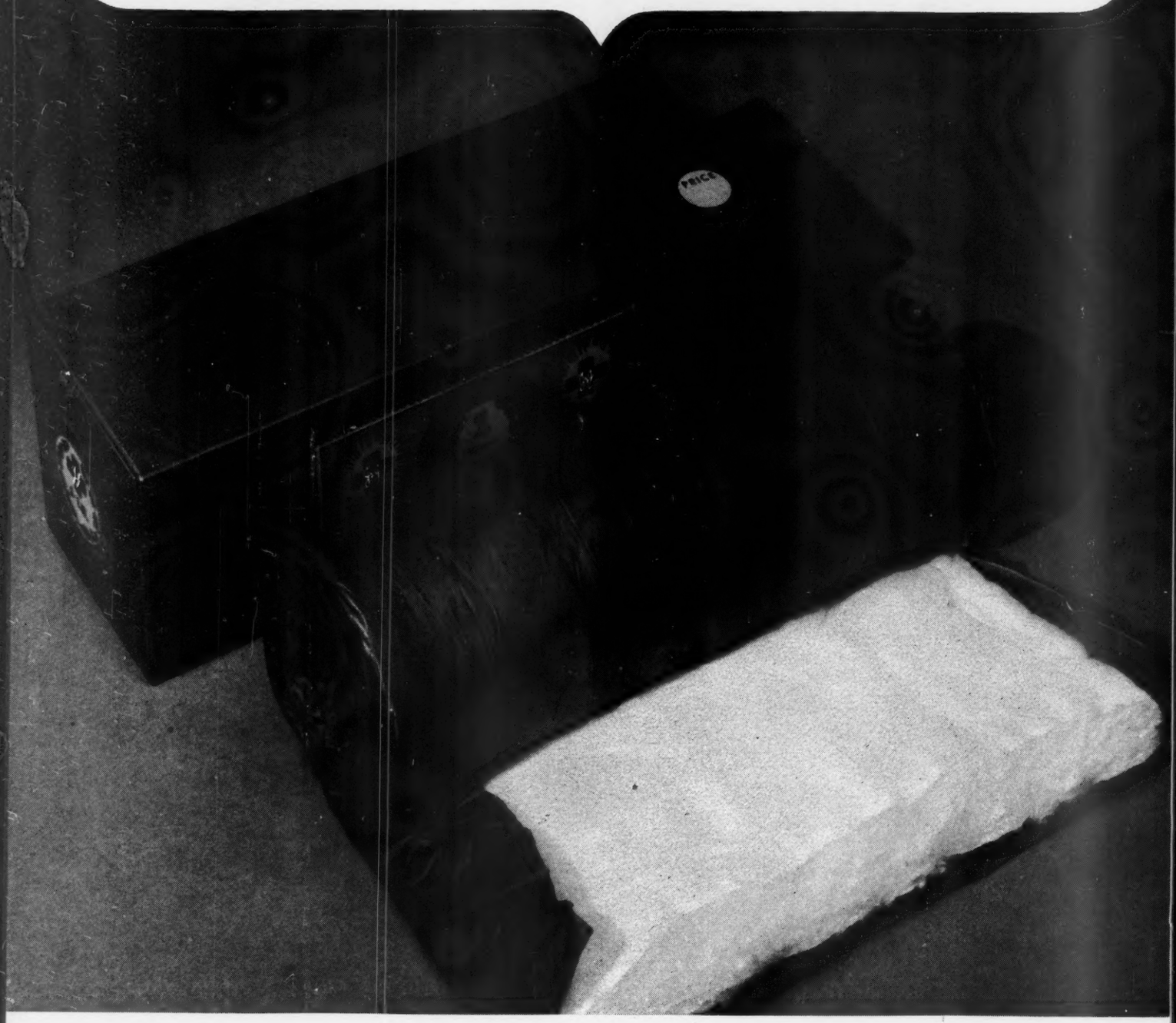

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Fifty-fifth Year of Continuous Publication

VOL. LXXI APRIL, 1946 NUMBER FOUR

Editorial

THE AMERICAN MEDICAL ASSOCIATION SHOWS THE WAY

TO paraphrase Winchell, "Orchids to the American Medical Association for their sane and straightforward blueprint of prepayment sickness insurance plans and health program." Many physicians, from general practitioner to full time professor, believe we have turned a corner and are witnessing the birth of a new era in the economic life of our profession. After long study and consideration a series of meetings were held in Chicago (beginning February 13 and the Board of Trustees and the Council on Medical Service "completed consideration which made possible a long step toward protecting the American people against costs of sickness."

Dr. Morris Fishbein, in an editorial (February 23, 1946) in The Journal of the American Association wrote:

"The fundamental step in the development of this plan was the establishment of standards of acceptance for medical care plans which have the approval of the Council on Medical Service of the American Medical Association. Any plan which meets the standards of the Council will be entitled to display the seal of acceptance of the American Medical Association on its policies and on all of its announcements and promotional material. In order to qualify for acceptance, the prepayment plan must have the approval of the state or county medical society in the area in which it operates. The medical profession in the area must assume responsibility for the medical services included in the benefits. Plans must provide free choice of a qualified doctor of medicine and maintain the personal, confidential relationship between patient and physician. The plans must be organized and operated to provide the greatest possible benefits in medical care to the subscriber.

"Medical care plans may be in terms of either cash indemnity or service units, with the understanding that benefits paid in cash are to be used to assist in paying the costs incurred for medical service. The standards also include provisions relative to the actuarial data that are required, systems of accounting, supervision by appropriate state authorities and periodic checking and reporting of the progress of the plan to the Council.

"Coincidentally with the announcement of these standards of acceptance there was organized, as a voluntary federation, an organization known as Associated Medical Care Plans, Inc. This independent association will include as members all plans that meet the minimum standard of the Council on Medical Service of the American Medical Association. The Associated Medical Care Plans will undertake to establish

coordination and reciprocity among all of these plans to permit transference of subscribers from one plan to another and use of the benefits in any state in which a subscriber happens to be located. Under this method great industrial organizations with plants in various portions of the United States will be able to secure coverage for all their employees. Moreover, it will be possible for the Veterans Administration, welfare and industrial groups and government agencies to provide coverage for the people in any given area through a system of national enrolment. In addition the Associated Medical Care Plans, Inc., will undertake research and the compliation of statistics on medical care, provide consultation and information services based on the records of existing plans and engage in a great campaign of public education as to the medical service plan movement under the auspices of state and county medical societies.

"The Board of Trustees of the American Medical Association also announced establishment of a Division of Prepayment Medical Care Plans with a director and a staff who will administer the activities related to the promotion and development of medical care plans in all the states.

"In announcing these proposals for a nationwide provision of sickness insurance on a mutual nonprofit basis, the Board also presented a complete health program with ten points, which include the development of services in the field of preventive medicine, maternal and child health, voluntary prepayment plans for protection against the costs of sickness, compensation for loss of wages due to illness, the care of the veteran and the development of a high standard of housing, nutrition, clothing and recreation."

Many informed physicians believe the prepayment sickness insurance plans will succeed. Surely it is the American Way. Once it becomes established and is a part of our professional life the nightmare of some form of socialized medicine with a crew of rapacious politicians at the helm will fade and die.

We hope those who read these lines or who have read the original editorial in The Journal of the A. M. A. will give the proposals thought and study, and then go forth and "preach the gospel" so that these sane, practical and forward-looking plans will become a reality.

Again, to those gentlemen of The American Medical Association who have labored for the good of the profession as a whole, we make a deep and sincere bow.

T. S. W.

Original Articles

URINARY CALCULI IN RECUMBENT FRACTURE PATIENTS

CAPT. HERBERT C. FETT AND COMDR. JOHN T. KANE
MEDICAL CORPS, UNITED STATES NAVAL RESERVE

THE presence of urinary calculi in fracture cases attracted the attention of the authors during the past year because of the extreme discomfort to the patient already disabled, and because of residual damage to the kidney which sometimes necessitates its removal.

On an active orthopedic service of about 700 beds, it was our privilege to observe twelve cases of fracture with this complication. All cases were examined and treated by the Department of Genito-urinary Surgery, while the fracture treatment was continued. These cases are herewith reported in the hope that some light may be thrown upon the prevention and treatment of this rather common complication of fracture patients who are kept recumbent over a long period of time.

LITERATURE

Lediard (1875) recorded a case of stone in the bladder developing after treatment for a spinal fracture in which the urine was heavily infected. Wilson (1931) reported instances of renal colic and hematuria in twenty-three out of 150 men who suffered from fractured femurs during World War I. His patients were recumbent for periods of from three to twelve months, and most had severe wounds, often infected, which were sometimes associated with ununited fractures. Hematuria was present in nearly every case and all had renal colic, which was sometimes bilateral. The symptoms usually occurred on the first day the patient was allowed out of bed. One patient passed three or four friable calculi. In all but two the urine was sterile. A few patients only were *x*-rayed, and in these, no calculi were demonstrated. Paul, and also Borman, recorded some cases of "recumbency calculi" developing in patients suffering from bone suppuration. Wallenstein (1931) recorded a case of bilateral renal calculi, one of which was demonstrated radiographically five weeks after immobilization for a fractured spine with paraplegia for which laminectomy was performed; the urine was probably infected, as regular catheterization was carried out for a period. Mawson (1932) recorded twenty-five cases of urolithiasis in children between the ages of eighteen months and fourteen years who were immobilized for tuberculosis of the spine or hip, slipped epiphysis, septic arthritis of the hip and knee, and spina bifida. Brown and Earlam reported six cases of "recumbency calculi." They divide calculi of this class into those which arise in association with a non-infected lesion, such as a fractured pelvis and those arising in patients suffering from bone suppuration. Holmes and Caplan (1934) recorded cases of extensive bilateral calculus formation rapidly developing after a fractured spine with paraplegia. McCague (1905) recorded ten cases of calculi following fractured pelvis, osteomyelitis of the femur, shell injury, and fractured spine, in four of which cases the calculi were bilateral. Key (1936) investigated complications in the urinary tract in 200 children recumbent for various orthopedic diseases and found such complications in thirty-two cases, mostly children

under treatment for tuberculous hips or spines. In twenty out of thirty-two instances, calculus (renal, ureteric, or vesical) was present, pyelitis in nine, and renal tuberculosis in three. Sick records five cases of recumbency calculi following fractured pelvis or spine.

ETIOLOGY

I. *Anatomy of Urinary Tract Favors Stasis and Infection When the Patient Is in the Dorsal Decubitus Position.* The hilum of the kidney faces forward and inward; in the dorsal decubitus the renal watershed will, therefore, lie at the pelvi-ureteral junction resulting in a tendency for a reservoir to form in the calici pelvic system. There is consequently more time for salts in a concentrated urine to pass out of solution—salts that would otherwise not have been deposited until the urine was voided. In addition, there is a gravitational influence upon any cells or débris, which may cause them to remain as potential stone nuclei.

In its early stages, the renal calculus of the ambulatory patient is usually located in the lower calices or pelvis, and is very rarely found in the upper calices; this is probably due to the fact that, in the erect position, the lower calices lie below the outlet of the renal pelvis, and are, therefore, less perfectly drained than the remainder of the system. In the recumbent patient, however, the calices are all placed under approximately the same conditions of drainage, and the calculus is frequently found in the upper, as well as in the middle and lower calices.

II. *Stone Areas.* According to Lassen, there is some predilection of the condition for so-called stone areas. In Africa, urinary calculi are frequent among the population of the Northern and Eastern coasts. Also, in Dutch East Indies, South China, the Valley of the Volga River, Mesopotamia, Persia, Afghanistan, Turkestan, Northwestern India and Japan. Our patients were all young men whose homes were scattered throughout the United States and had seen service in both the Atlantic and Pacific areas.

III. *Dietary Effects.* In many children's orthopedic hospitals, there is a tendency to emphasize the ingestion of large quantities of milk. The excess calcium intake in conjunction with the hypercalcemia normally associated with fractures and other cases of bone disorders causes further excretion of calcium in the urine. The latter combined with the poor drainage from the kidney because of the patient's recumbency may cause a precipitation of calcium. Dehydration resulting from insolation and low intake of fluid are also contributory to calculi formation. In most instances in which calcium stones have been formed, the precipitation took place in an alkaline medium.

IV. *Heredity.* This seems of actual significance among persons who have cystinuria or cystin-calculi, which conditions are decidedly familial in nature. In our series there was no family history of urinary calculi.

V. *Infection.* Infection seems to have the effect of changing the reaction of the urine and particularly the effect of increasing the hydrogen ion concentration. As a consequence, the condition of saturation in the urine often will undergo considerable change, so that the possibility of precipitation, especially of phosphates and carbonates becomes greater. This is seen in particular in cases of infection by urea-splitting organisms, members of the genus Proteus and others. It is possible that in the case of infection, there is also an increase in colloids, or that colloid metabolism is changed. Theoretically, as the colloids split, more surface may be present, and adsorption may take place. However, colloidal suspensions are rarely stable because on standing, the particles tend to coalesce and reduce the surface area. Furthermore, bacteria and inflammatory products themselves may form nuclei for stone, which may become incrusted and continue to grow. Rovsing, Hellstrom and other authors, have suggested such a pos-

sibility. Priestley, also, reports certain bacteria such as proteus ammoniae, staphylococci, and certain streptococci which to such deficiency being a contributory factor in the formation of renal calculi in general. Mawson points out that many

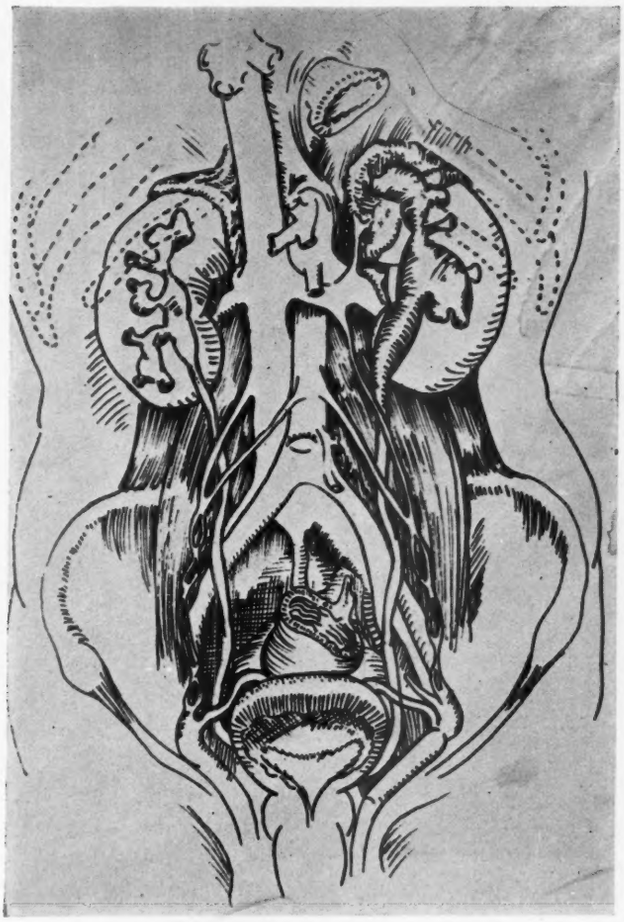

FIG. 1. Anatomy of the urinary tract.

split urea into ammonia and carbon dioxide, thereby creating an alkaline urine. These are the organisms most commonly associated with the presence of stones. Bacillus proteus is due to contamination. If an individual has calculi entirely removed, he may have further calculi precipitated if Proteus infection continues.

VI. *Hyperparathyroidism.* Interest in the dysfunction of the parathyroid glands has been stimulated lately. However, most authors agree that cases of urinary calculi due to disease of the parathyroid glands are rather rare. Barney and Mintz state that hyperparathyroidism is responsible for about 1 per cent or less.

VII. *Deficiency of Vitamin A.* The evidence so far available points strongly parts of the world where renal calculus is very common, the diet consists largely of carbohydrate foods and is deficient in proteins and in vitamin A. The diet containing vitamin A is usually adequate in most hospitals, but the power of absorption of vitamin A by the bowel may be defective in some cases.

VIII. *The Possibility of Hypervitaminosis D Resulting in Renal Calcification.* Experimental work with rabbits, dogs and guinea pigs seems to demonstrate some relationship. Thatcher (1936) has recently recorded a further case of a baby eleven and one-half months old, who died after a period of failing health of four months. The only cause of death found at the autopsy was calcification in the kidneys

resembling exactly the changes found in fatal cases of hypervitaminosis D reported in animals. We are on more difficult ground when we come to the question of the overproduction of the vitamin D by insolation. Vitamin D is synthesized in the skin by the action of ultra violet light. Pigmentation constitutes the natural defense mechanism of the body against this overproduction by the action of the sun's rays. Any marked excess of vitamin D in the body will result in the elevation of the serum calcium. Hoyle suggested that prolonged exposure to the sun may be a cause of stones in the kidney. However, most authors at present, feel that the elevated calcium in the blood is due to the hypercalcemia normally associated with fractures.

IX. *Congenital conditions* such as horseshoe kidney, aberrant vessels, valvular formations in the pelvis of the kidney or ureter, strictures and the like, or acquired lesions, such as infection of the urinary system by various bacteria, ren mobilia,

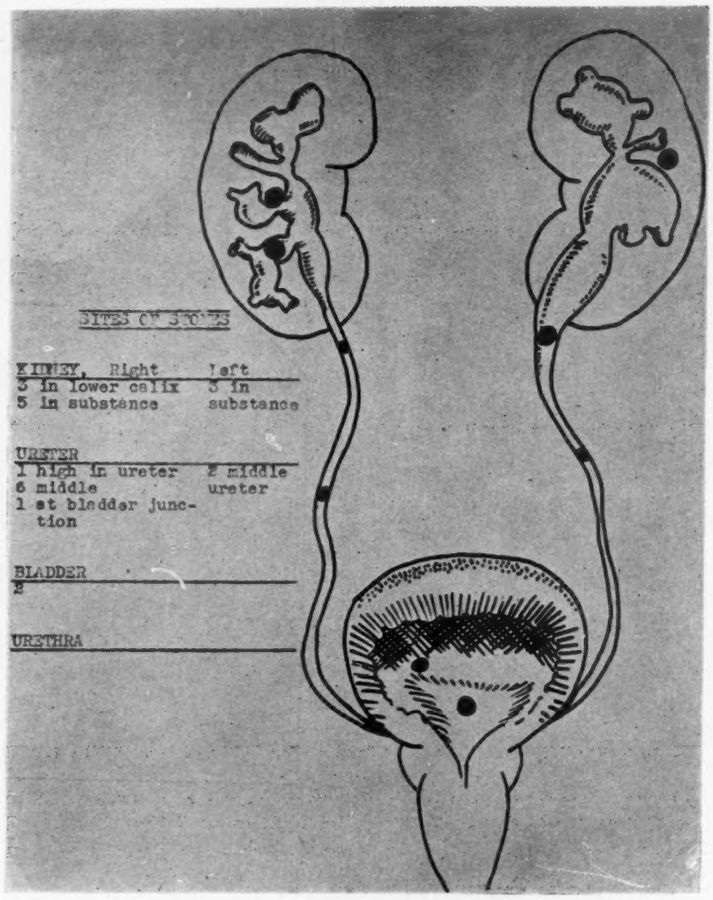

Fig. 2. Sites of urinary calculi.

stricture and others, may be contributory. There was one case in our group of stricture of urethra as an added complication of simple fracture of the first and second lumbar vertebrae.

Flocks' most recent article on calcium urolithiasis mentions the following conditions which predispose to urinary stone.

I. Disease producing prolonged immobilization of the body, such as,

(a) Fractures of the spine or extremities associated with prolonged immobilization of long bones.
(b) Chronic osteomyelitis
(c) Chronic arthritis, or other bone joint disease producing immo-

bilization of large portions of the skeleton.

(d) Neurological damage as result of trauma-producing immobilization.

II. Changes in Urinary Organs:
(a) Congenital anomalies associated with stasis
(b) Acquired obstruction such as stricture of urethra
(c) Paralysis of urinary passageway
(d) Introduction of infection into urinary tract
(e) Foreign body in urinary passageway

III. Endocrinopathies:
(a) Hyperparathyroidism

IV. Forms of infection elsewhere in body?

V. Vitamin deficiency or excess:
(a) Vitamin A deficiency
(b) Vitamin D excess

VI. Metabolic Abnormalities:
(a) Idiopathic hypercalcaemia
(b) Changes in colloids

INCIDENCE

The urinary calculi to be discussed occurred in white males between the ages of twenty and thirty, average twenty-five plus. In our group of twelve cases it was of interest to note there were no negroes. In 150 war cases with fractures of the femur, Wilson, in England, reported 15 per cent with renal colic and hematuria. Cases have been reported as early as eighteen months of age. The condition occurs more frequently in the male than in the female. This ratio is almost four males to every three females as reported by English study. The prostate gland is said to be responsible for the difference between the ratio of the male and female. It is easily understood that with hypertrophy, infection or new growth, the prostate may so constrict the urethra that the bladder, ureters, and kidneys will not be able to drain properly. This static condition of urine is again conducive to precipitation, especially if there is an associated urinary infection.

SYMPTOMATOLOGY

The symptoms are the same as those seen with calculi in non-recumbent patient, that is, colic, blood in the urine, and often the passage of the calculus in a few days. According to Carlson and Ockerblad, it occurs more frequently when the patient's position is changed after being recumbent for a long period of time. The diagnosis is substantiated by *x*-ray examination. Since most of the stones are calcium stones, even the earliest calculi may be seen on a good *x*-ray plate. According to Pyran and Fow Weather, hematuria is the foremost symptom in urinary calculi associated with recumbency. However, in urinary stones associated with enforced recumbency, experience shows colic is more common than is hematuria. In some few cases, calculi manifest their presence by the sudden appearance of chills, and fevers of unknown origin. This, of course, is indicative of associated infection and microscopic examination of the urine will make the diagnosis. It must be remembered, however, that a true pyonephrosis may be present in conjunction with renal stones and the urine will give no hint of renal infection.

In Order of Appearance	No. of Cases
Colic and pain	10
Hematuria	6
Burning on urination	3
Costo-Vertebral tenderness	3
Frequency	1
Chill	1

Note: Some of the patients manifested more than one symptom.

SIGNS

The renal calculus, without infection, does not afford diagnosis by any physical diagnostic sign. However, when obstruction occurs, the usual sign of renal pelvic distention is present—tenderness to deep pressure at the costal vertebral angle of the affected side. The abdomen even during accute attacks of renal colic, may or may not reveal evidence of muscular spasticity. When there is no spasticity the patient will often complain of a generalized abdominal

Urine Reaction	Injury	Crystals	WBC in Urine	Number of Days of Immobilization	Type of Immobilization	Warning Signaling Investigation	Sulpha Therapy	Area from Which Admitted	Site of Stone	Other Complications	Type of Individual	Operations
Alkaline	Fracture Right Femur	Few dicalcium Phosphate	3–4 Leucocytes. Countless Lymphocytes	290	Plaster spica with pin incorporated	1. Rt. lumbar spasm 2. Burning on urination 3. Hematuria 25 days. 35 days (ambulant)	Yes	North Atlantic Area	Right ureterovesical junction	1. Infection of pin site 2. Another calculus is present	Tall and Thin	Bone graft and cystoscopic
Alkaline	Fracture Multiple Pelvis. Dislocated Lip Fracture left Femur and Acetabulum	Rare Calcium Oxalate. Rare Sulpha Crystal	2–3 h.p.f.	300	Roger-Anderson splint	1. Pain left kidney region	Yes	South Atlantic Area	Lower calix Right kidney Lower left ureter	Fracture Tibia and Fibula	Tall and Thin	Lane plate tibia closed Red. Femur
Acid	Fracture comp. Left Astragulus. Fracture comp. Left Femur	Much amorphous material. Many sodium urate crystals	Countless	270	Double Hip Spica	1. Pain burning lower left quadrant 2. Tenderness over Right Kidney	Yes	North Atlantic Area	Lower right Calices	None	Short and Stocky	Triple arthrodoesis Lane Plate Femur
Alkaline	Comp. Frac. Rt. Femur Draining wound over R. Patella	Calcium oxalate and carbonate	6–8 W.B.C.	76	Double Hip Spica	1. Pain and chill llq following urination	Yes	Pacific Area	Pelvis of left Kidney	Osteomyelitis	Tall and Thin	Sequestrectomy
Weakly acid	Comp. Frac. Rt. Femur Rt. Peroneal Nerve Injury	Calcium phos. oxalate	45 w.b.c.	517	Roger-Anderson splint Haynes splint	1. Pain in lower left quadrant	Yes	Pacific area	Small stone in each kidney & large one in left ureter	1. Abscess at pin site 2. Large stone still present	Stocky	Ostectomy Bone Graft Femur
Alkaline	Un-united frac. Left pubis and Ischium. Rupture Urethra	Triple and amm. phosphate	Countless	180—1st stone 270—2nd stone	Double Hip Spica	1. Pain in right lumbar region 2. Hematuria 3. Burning on urination	Yes	Pacific Area	1 in rt. pelvis and 3 in right lower ureter	Recurrence of calculi residualstricture Pelvic-Osteo	Tall and Thin	Plastic on urethra Cystoscopic
Alkaline	Fracture comp. Rt. Tibia and Fibula and Femur	Magnesium and calcium carbonate. Few calcium oxalate	Countless	223	Double Hip Spica	1. Pain in rt. loin radiating to groin 2. Costo-vertebral tenderness 3. Frequency	Yes	Pacific Area	Stone in rt. lower ureter	Osteomyelitis	Tall and Thin	Sequestrectomy

Urine Reaction	Injury	Crystals	WBC in Urine	Number of Days of Immobilization	Type of Immobilization	Warning Signaling Investigation	Sulpha Therapy	Area from Which Admitted	Site of Stone	Other Complications	Type of Individual	Operations
Alkaline	Comp. Frac. Rt. Femur Lesion of rt. Sciatic	Rare calcium oxalate	4–6 w.b.c.	128 316	Double Hip Spica	1. Pain in right lumbar area 2. Tenderness over rt. kidney 3. Hematuria	No	North Atlantic Area	Inferior rt. kidney pelvis Rt. ureteralstone (calix) Several right renal stones	Draining sinus at wound site	Stocky	Ostectomy and Bone Plated
Alkaline	Fracture simple and dislocation of 12th D vertebrae	Triple phosphates	Countless	622	None	1. Pain right costo-vertebral junction 2. Hematuria	Yes	Atlantic Coast	Right ureter bladder	Paraplegia and cord bladder	Tall and Thin	Cystotomy and Laminectomy
Weakly acid	Frac. Comp. Rt. Tibia	Triple Phosphates	4–6-w.b.c.	89	Cast right mid-thigh to toes	1. Pain left costo-vertebral area 2. Hematuria	Yes	Pacific Area	Pelvic portion of left ureter	Draining sinus right leg	Tall and Thin	Debridement
Weakly acid	Simple Frac. of L-1 & L-2 Lacerated left hand	Much Amorphous material	Countless	471	Plaster Jacket	1. Pain over bladder and burning on urination	Yes	North Atlantic Area	Calculus over trigone of bladder	Stricture of urethra and swollen right testicle	Tall and Thin	Laminectomy Cystotomy
Alkaline	Compression Fractures of D-3 & D-4 Frac. rt. Femur and acetabulum	Triple Phosphates	2–4-w.b.c.	113	Plaster Jacket	1. Hematuria	Yes	California USA	No IVP performed at time stone passed	Paraplegia still	Tall and Thin	Laminectomy
7 cases Alkaline 4 cases weakly acid 1 acid			Low 76 days High 622 days Av. 276 days			1. Pain outstanding complaint in 10 cases 2. Burning on urination 3. Hematuria 6 cases	11 Patients had Sulpha	6 Atlantic 5 Pacific 1 U. S.				

tenderness upon deep palpation. On the basis of Flocks classification, much can be learned from the cases shown in the chart below. The following twelve cases are those with fractures complicated by urinary calculi:

Certain pertinent facts are immediately obvious upon examination of the above cases:

1. In seven of the twelve cases, the urine was definitely alkaline; in four cases it was weakly acid, and in one instance, it was noticeably acid.

2. Examination of the crystals of the urine indicate that triple phosphates were present in four cases, which indicates an infectious process. Six cases demonstrated calcium oxalate, calcium carbonate, and phosphate. The other two cases demonstrated either oxalate or urate crystals.

3. Examination of the white blood cells in the urine revealed that in only six instances was the pyuria marked. In one of this group, the calculi were bilateral. The remaining six cases were not impressive in their urinary white blood count. Our interpretation of the latter fact, is that urinary stones may occur in the absence of infection in the urinary tract.

The number of days of immobilization prior to the appearance of kidney stones is somewhat variable. The shortest time for the occurrence of urinary stones was seventy six days, and the maximum time for the occurrence of urinary stones was 622 days after fracture. The group of twelve cases averaged 276 days. Another factor, which is of interest, is that no stone occurred when the individual became ambulatory. All stones were manifest while the patient was still recumbent. In an examination of the symptoms, pain was the outstanding complaint in ten of the cases. The second most frequent symptom was hematuria, which was macroscopic in six of the cases. The third outstanding symptom was burning on urination. A chill was present in only one of the twelve cases reported. This patient's urine, however, was not infected. Urinary stones occurred on the right side fifteen times and five times on the left side, with the two remaining stones in the trigone of the bladder, and the ureterovesical junction. Sulfa therapy was administered in eleven of the twelve cases. However, no sulfa stones were discovered in this series.

Six of the cases were from the Atlantic area, five from the Pacific, and one from California. This would tend to indicate that the climatic condition does not play a major part in the formation of urinary calculi.

Examination of the sites of the calculi would seem to indicate that in ten instances the process was early because of the fact that the stones were found in the calices, pelvis, or upper ureter. In only two cases were these stones found in the bladder. Such high calculi should be more amenable to therapy.

The individuals as a group were classified as either thin or stocky. Three of the group were stocky, and the other nine were thin. No importance has previously been attached to this fact but when it is recalled that nephroptosis is more common in the tall and thin individual such a point may be worthy of further investigation.

TREATMENT

It is the opinion of the authors that the therapy and prophylaxis should be directed toward:

1. Improving the drainage of the kidney by daily changes of posture, by exercising the uneffected parts, and keeping infection at a minimum. With the personnel provided by the physical training program of the Navy it has been no problem to maintain the muscle tone and thereby provide adequate circulation.

2. Diet is considered to be of some significance. A high acid ash diet maintains the urine in an acid state and prevents the formation of stones which occur in an alkali media.

3. In the event that the urine still remains alkaline, it is suggested that ammonium chloride 8 to 10 gr. three times

a day be given to supplement the acid ash diet. It is recommended that the hydrogen ion concentration be maintained at 5.5. This may be controlled by daily urinalysis.

4. Fluids should be forced at all times in order to prevent stasis and to minimize the dehydration which occurs in hospitals in winter time, and outdoors in the summer time.

5. Every effort should be made to combat staphylococcic, streptococcic and proteous infection.

6. Sulfathiazole and sulfadiazine both may be used to combat infection as the case warrants. Penicillin is of no use in strictly Proteus infection.

7. Catheterization should be discouraged, as not infrequently, it will result in an ascending infection of the genitourinary tract.

8. Monthly flat plates should be taken of the abdomen to detect early calcification in kidneys and bladder.

CONCLUSION

A discussion has been made of twelve cases of urinary calculi which complicated fractures. An effort to point out the pitfalls which may be encountered was made, in order to anticipate and to treat patients with this misfortune. The advent of such appliances as the Haynes' Splint, Stader Splint or Roger Anderson or other apparatus which facilitates early activity and minimal recumbency should further assist in preventing stasis and prolonged fixation of the extremities. It is hoped by the authors that the above paper may be of some assistance in preventing the occurrence of recumbency calculi.

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SPLENECTOMY FOR TRAUMA

PRACTICAL POINTS IN SURGICAL TECHNIC

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SPLENECTOMY is not a common operation in temperate climates. Partly because of this, and partly because it sometimes is a difficult and hazardous surgical procedure, it is a favorite choice of senior surgeons and professors of surgery. Consequently, it is not uncommon, even in the larger clinics, for well qualified surgeons to complete their training with little or no personal experience with this operation. While it may be a sound policy to concentrate the surgery of those spleens that are greatly enlarged, or are associated with certain blood dyscrasias, in the hands of the highly experienced few, the removal of the ruptured spleen is an operation which must, perforce, be performed by the comparatively inexperienced man. This is particularly true of military surgery, in which injuries of the spleen are encountered more frequently than in civilian practice.

The removal of the ruptured spleen is not a difficult procedure if certain anatomical facts are appreciated by the surgeon. This presentation of the technic has been prompted by the sight, on more than one occasion, of surgeons of considerable experience struggling in the depths of the abdomen in an effort to secure the entire gastrosplenic and lienorenal ligaments in a single pedicle clamp, and by the realization that a number of able surgeons appear to be unaware of the basic contributions of Balfour[1] to this field.

SURGICAL ANATOMY

The splenic pedicle is composed of three separate layers well described by Henry[4] as an "anatomical sandwich." An understanding of the nature and relationship of these layers is an essential prerequisite to a well ordered splenectomy. From before, backward, these are: (1) *The presplenic fold*, a thin, double peritoneal fold extending from the great omentum to the lower pole of the spleen. (Fig. 10.) In the small, previously normal spleens of acute trauma, it is an inconsequential structure often divided under the mistaken notion that it is a peritoneal adhesion. It may contain a few small veins. In enlarged spleens this layer may become a greatly thickened and formidable barrier;[3] (2) the *gastrosplenic ligament*, a double fold of peritoneum containing branches derived from the left gastro-epiploic artery and vein. This fold extends from the stomach to the spleen, to which it is attached along the anterior margin of the hilum from upper to lower pole. It holds the stomach in close proximity to the upper pole of the spleen; (3) *the lienorenal ligament*, a double fold of peritoneum containing the splenic artery and vein and the tail of the pancreas. These layers are represented diagrammatically in Figure 1A.

Approached from in front, the entire pedicle, with its three layers and five peritoneal reflections, lies between the operator and the splenic artery, which in the case of traumatic rupture of the spleen is the immediate goal. From behind, however, only a single peritoneal covering and the tail of the pancreas intervene between artery and surgeon. Actually, in the small, previously normal spleens encountered in acute trauma this posterior reflection is a delicate structure often torn in delivering the spleen, and the pancreas rarely extends to the end of the pedicle. So once the spleen is delivered and the pedicle viewed from behind, the splenic artery is completely exposed and can be secured at once. This fragment of surgical anatomy was pointed

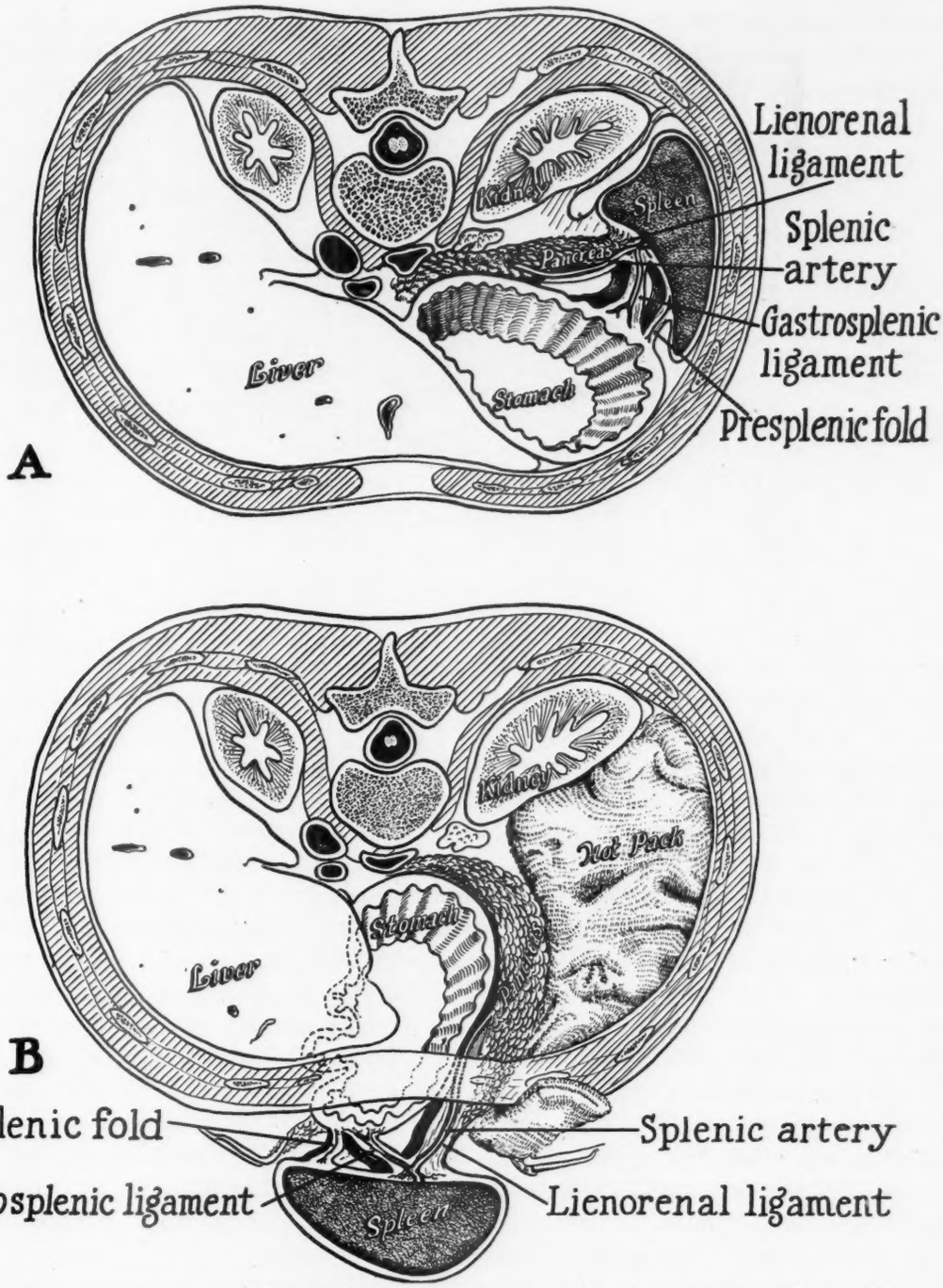

Fig. 1. A, diagrammatic representation of the surgical anatomy of the spleen. The three layers of the splenic pedicle bar the surgical approach to the splenic artery from in front. B, diagrammatic representation of the anatomical relationship after delivery of the spleen and insertion of a gauze pack. From behind only the tail of the pancreas and a thin peritoneal covering intervene between artery and surgeon. Actually, as described in the text, in most instances the artery is completely exposed in its distal portion by this maneuver.

out and beautifully illustrated by Balfour over two decades ago.[1] It is illustrated diagrammatically in Figure 1B.

torn completely from its pedicle, this route may be the most expeditious way of securing the retracted bleeding vessels.

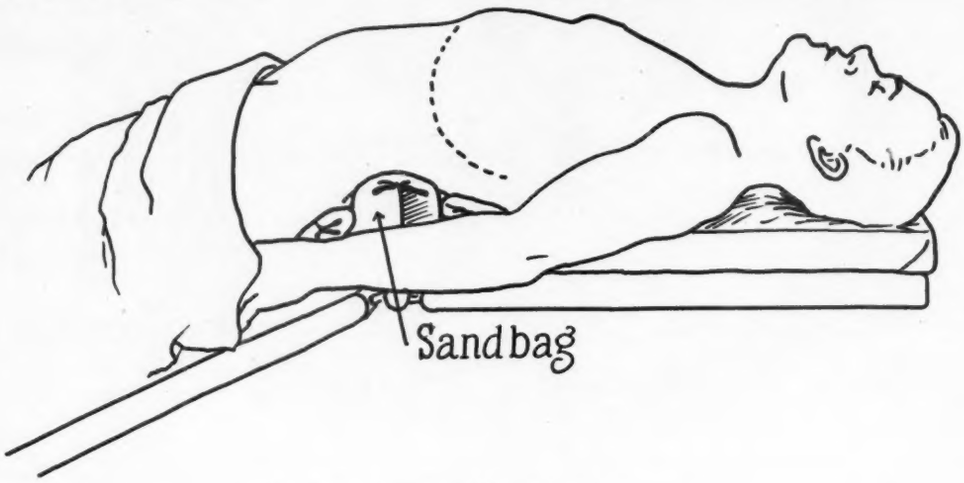

FIG. 2. Optimum position for splenectomy.

Four attachments may interfere with easy mobilization of the spleen; (1) The posterior layer of the lienorenal ligament may be fused retroperitoneally; (2) the upper fused edge of the gastrocolic and lienorenal ligament may be attached to the diaphragm (phrenicosplenic ligament); (3) there may be adhesions between spleen and diaphragm; or, (4) between spleen and splenic flexure of the colon (lienocolic ligament). Although in certain cases of enlargement of the spleen these attachments may become quite vascular, normally they contain no major blood vessels.

One additional anatomical feature to be kept in mind is the long course of the splenic artery along the upper edge of the pancreas, which fact permits it to be exposed through the lesser peritoneal cavity some distance from the spleen. This is of considerable importance in dealing with very large spleens since it permits control of a major portion of the blood supply before attempting to mobilize the spleen.[2] This fact is not of great importance in the surgery of the ruptured spleen when the primary purpose is to secure the bleeding vessel, and the small size of the spleen makes delivery and exposure comparatively easy. Rarely, as will be pointed out later, in cases in which the spleen has been

DIAGNOSIS

There may be considerable variation in the severity of the symptoms, depending upon the degree of injury. If the trauma is sufficient to produce immediate rupture and the spleen is the only organ injured, the diagnosis is not difficult. A history of a blow on the flank, followed by pain in the left upper quadrant, later extending throughout the abdomen, increasing evidence of peritoneal irritation, pain in the region of the left shoulder, dyspnea, and progressive shock, are characteristic. However, a contusion of the spleen can be produced by injuries so trivial as to be forgotten by the patient, and actual rupture can be delayed for periods varying from several hours to as long as a week or ten days.

If there is an extensive contusion of the muscles of the flank and abdominal wall, multiple fractured ribs on the left, fractures of the transverse processes of the lumbar vertebrae, and a contusion of the kidney, it may tax the skill of the most experienced clinician to determine the extent of injury to the spleen. In such cases, careful, repeated examination of the abdomen is necessary to establish a diagnosis. Recurrence of shock after adequate re-

placement of blood, together with increasing evidence of peritoneal irritation or signs of shifting dullness in the abdomen may be the first conclusive evidence of an intraperitoneal injury. Diagnostic abdominal paracentesis has been recommended. This procedure is not without risk, and the results obtained by it are by no means entirely reliable.

PREOPERATIVE PREPARATION

The recorded mortality in rupture of the spleen varies between 30 and 40 per cent.[5] The amount of blood loss in these cases may be staggering and inadequate replacement is probably the most important single contributing factor to the high mortality. Adequate shock therapy is, therefore, the most essential preoperative preparation. Large amounts of whole blood are required to maintain a normal circulatory state. Whole blood, rather than, or in addition to plasma is vitally important. Complete reliance upon plasma as a replacement fluid in cases of severe hemorrhage in which extensive surgery is necessary has been singularly disappointing.[3]

One additional preoperative measure of considerable value, is to pass a Levin tube into the stomach and leave it *in situ* during the operation thus assuring that the operative field is not obscured by a large dilated stomach.

OPERATIVE TECHNIC

The optimum position of the patient is shown in Figure 2. The table is broken slightly at the level of the costal margin, anteriorly, and the body rotated slightly toward the right and supported in this position by a sand bag in the loin. This elevation on the flank permits a lateral T-shaped extension of the left rectus incision in difficult cases. (Fig. 3.)

The abdomen is opened through a long paramedian left rectus muscle-splitting incision. This should extend up to the costal margin and below to the umbilicus, or a little beyond. Many surgeons prefer an oblique incision for splenectomy, but in dealing with the ruptured spleen the left rectus incision is preferable because it

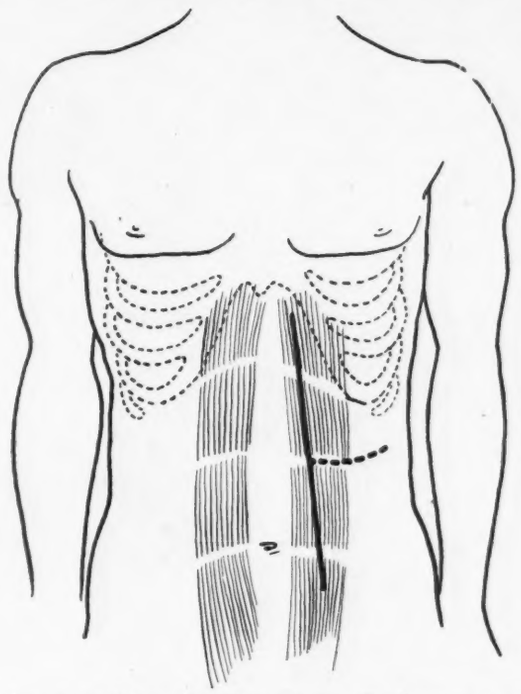

FIG. 3. Incision for splenectomy. Perference for the left rectus incision rather than an oblique subcostal approach is discussed in the text. The T-shaped lateral extension is rarely necessary.

can be made more quickly and easily, permits better exposure of the rest of the abdomen in case of associated injuries to the liver or intestines, and is entirely adequate for removal of the small previously normal spleen. Rarely, if the pedicle is lost, or the spleen torn completely from its pedicle, the T-shaped extension as shown in Figure 3, may be needed to provide more complete exposure. The transpleural approach to the injured spleen is recommended only when there is an associated injury to the chest which requires an immediate left thoracotomy.

Once the abdomen is opened, exploration completed, the diagnosis confirmed, and the immediate field cleared of blood, the spleen should be delivered into the wound and the bed firmly packed with a large, warm, moist gauze pad. In delivering the spleen it is useless to attempt to pull it out by the slippery lower pole. The hand must be advanced along the free lateral aspect

of the spleen gently separating diaphragmatic adhesions with the fingers. (Fig. 4, Insert A.) Once over the upper pole with the posterolateral margin free, the spleen should be *pushed* toward the wound. If the posterior layer of the lienorenal ligament is free, delivery is easier. If this layer is obliterated, further gently freeing with the fingers is necessary.

There is always a small amount of bleeding from diaphragmatic adhesions or retroperitoneal attachments after the spleen is delivered. As Balfour[1] has pointed out, the use of a huge gauze pack satisfactorily

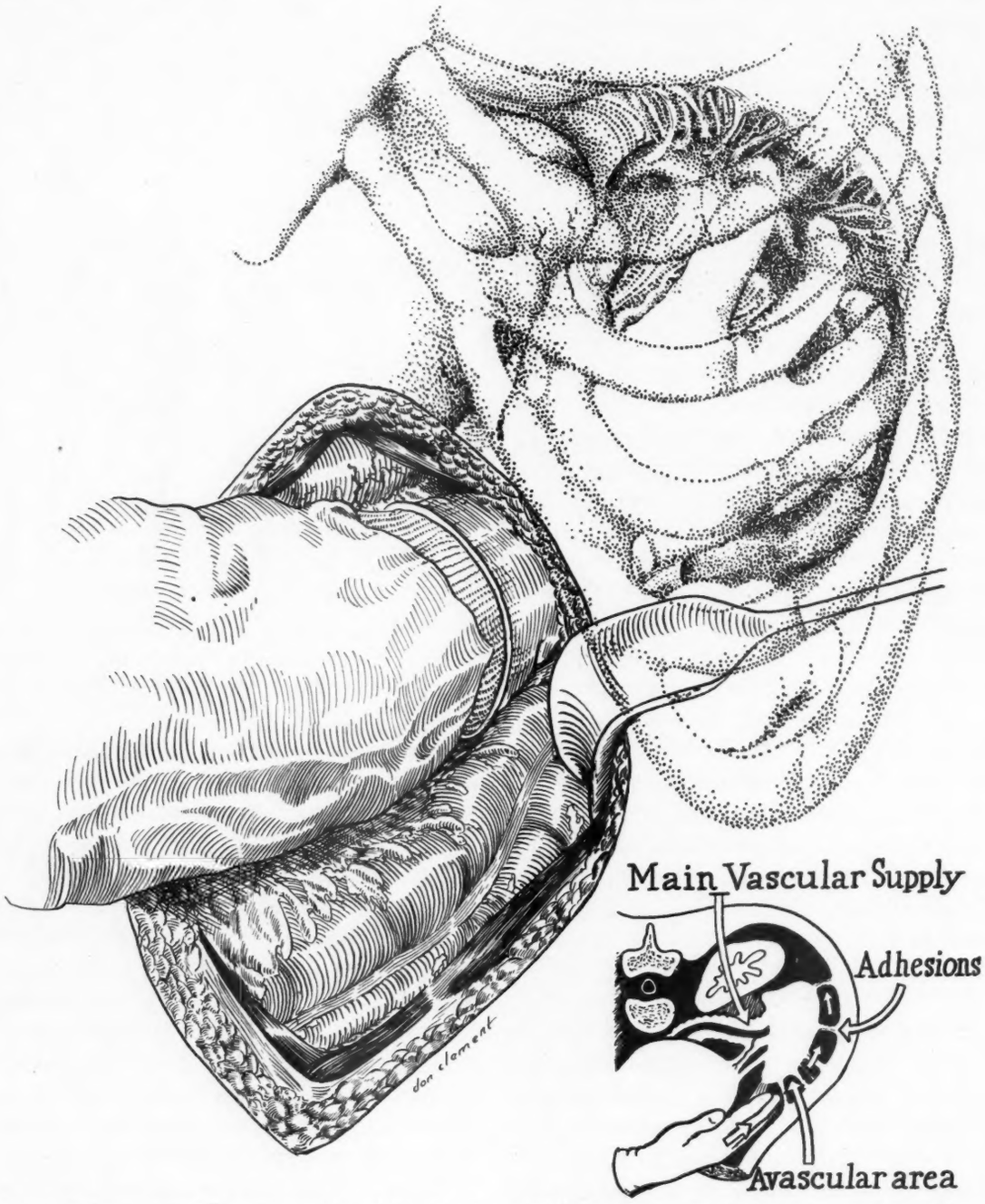

FIG. 4. Diagrammatic representation of the depth at which the spleen lies in relation to the incision. The operator's hand must reach above the upper pole and "push" the spleen down rather than try to grasp the tip of the lower pole. The insert shows points of fixation which may have to be gently separated by the fingers and the direction in which the hand should be advanced to avoid vascular areas.

controls this oozing even in the case of large adherent spleens. In the small spleens of acute trauma, therefore, no time need be lost in freeing the comparatively avascular adhesions or attachments which may be encountered. Usually, these can be gently separated by the fingers. At times it may be preferable to divide the posterior peritoneal reflexion with scissors, but this is time-consuming and usually not necessary. The lienocolic ligament is readily divided with scissors during or just after delivery of the spleen. This attachment of the spleen is easily overlooked however, and care must be taken not to injure the colon in freeing what appears to be just another adhesion.

How far distant the spleen lies in relation to the wound is shown diagrammatically in Figure 4. Despite this, the splenic pedicle is sufficiently mobile to permit delivery. The only danger is the possibility of tearing large veins in the lienorenal ligament, or pulling a badly fractured spleen completely away from its pedicle. This should rarely, if ever, happen if care is exercised. If these accidents just mentioned should occur, there is no cause for serious alarm as the situation can readily be controlled.

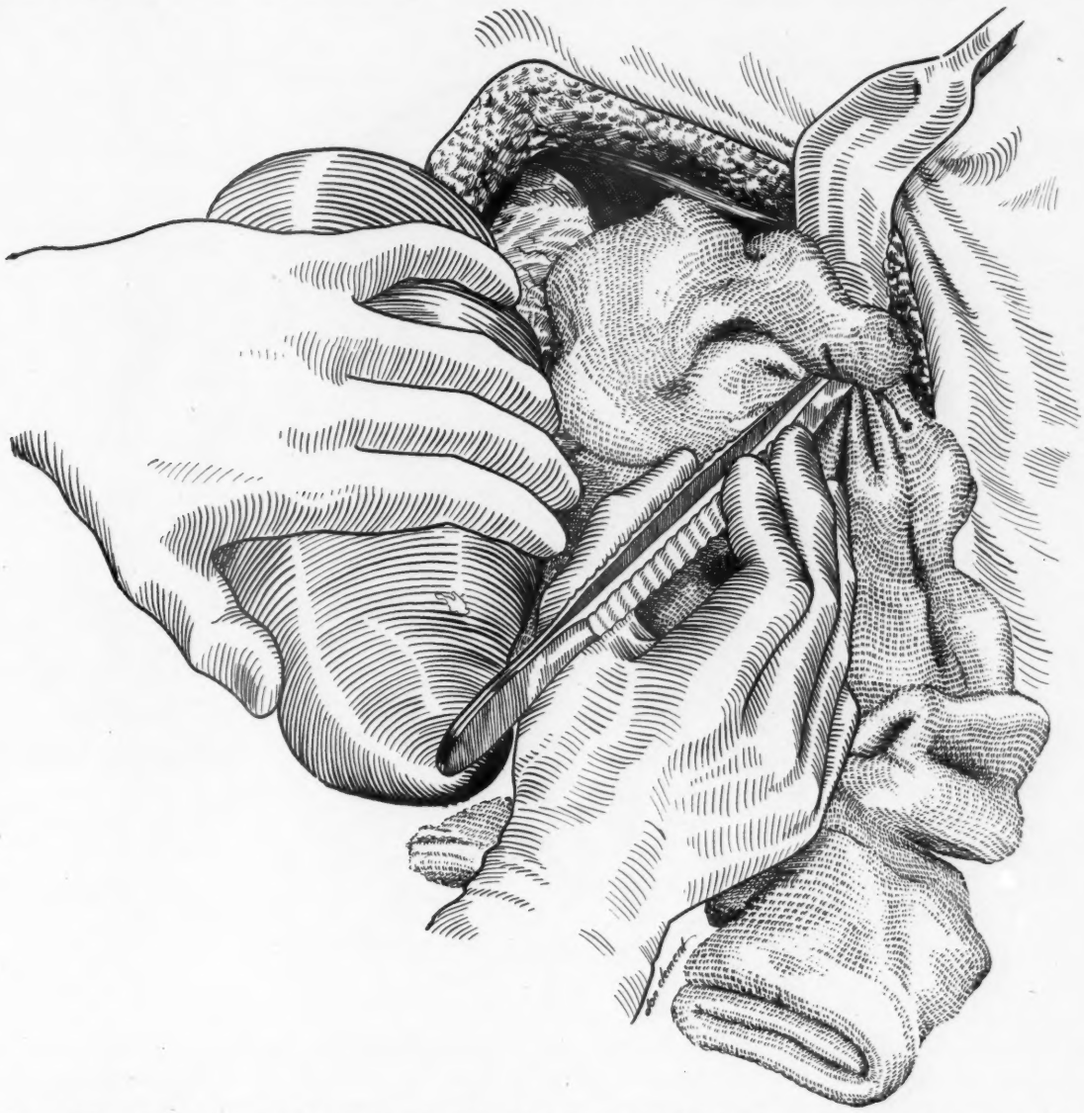

Fig. 5. The spleen is delivered and the splenic bed is being packed with a large, warm, moist gauze pad. This controls ozzing from the splenic bed and diaphragmatic adhesions. The final position of the pack is shown in Figure 1B.

As soon as the spleen is delivered it is held by the operator's left hand, and the cavity firmly packed with a large, warm, moist gauze pad. (Figs. 1B and 5.) Inspection of the posterior aspect of the pedicle will now reveal the splenic artery, or its major branches directly accessible. (Fig. 6.) These are secured at once with ligatures, and the remainder of the operation can be done quite leisurely. It should be noted that these vessels in the lienorenal ligament are isolated individually and tied under direct vision. No attempt is made to divide the vessels or pedicle at this point. Occasionally, in order to expose the artery it may be necessary to dissect the tail of the pancreas off the posterior aspect of the pedicle as indicated in Figure 6.

The spleen is now turned over to the left, exposing its anterior margin and the dissection of the presplenic fold and gastrosplenic ligament undertaken. (Fig. 7.) The former usually amounts to a mere wisp of tissue, and is easily severed. It may be absent. The latter requires division by careful isolation and ligature of vessels. With two fingers beneath the gastrosplenic ligament, separating it from the lienorenal ligament, it is very easy to isolate, clamp, and divide it in convenient bites which include major vessels, as shown in Figure 7. The proximity of the stomach to the

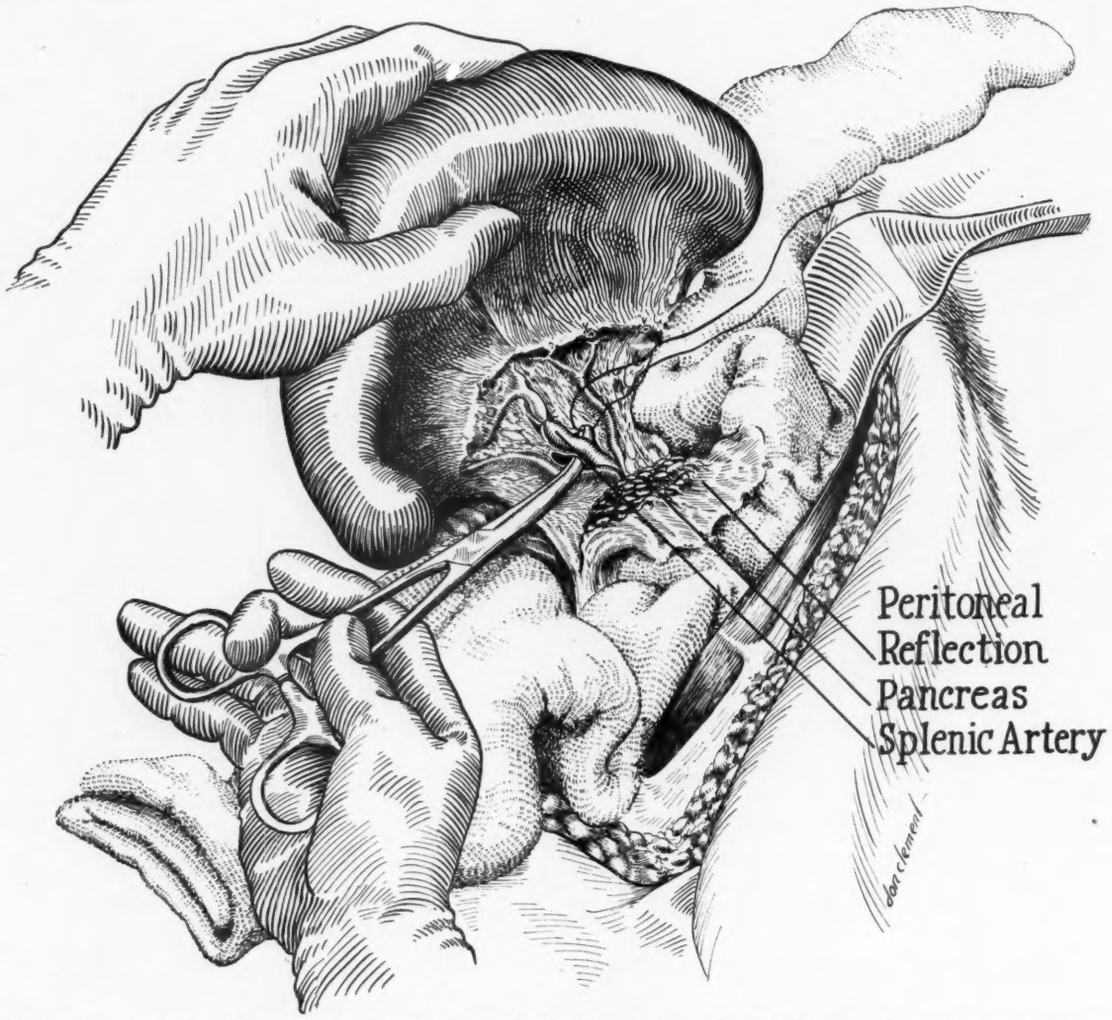

FIG. 6. The exposure of the splenic pedicle from behind. The peritoneal reflection is rarely as distinct a structure as it appears here. Also, it is rarely necessary to dissect the pancreas off the posterior aspect of the pedicle as it usually does not extend down as far as represented. However, access to the main branches of the splenic artery can be obtained as easily as represented.

spleen at the upper pole calls for this careful dissection of the gastrosplenic ligament. Actually, it takes only a few minutes to divided any attempt to clamp *en masse* the main pedicle is very likely to result in injury to the stomach.

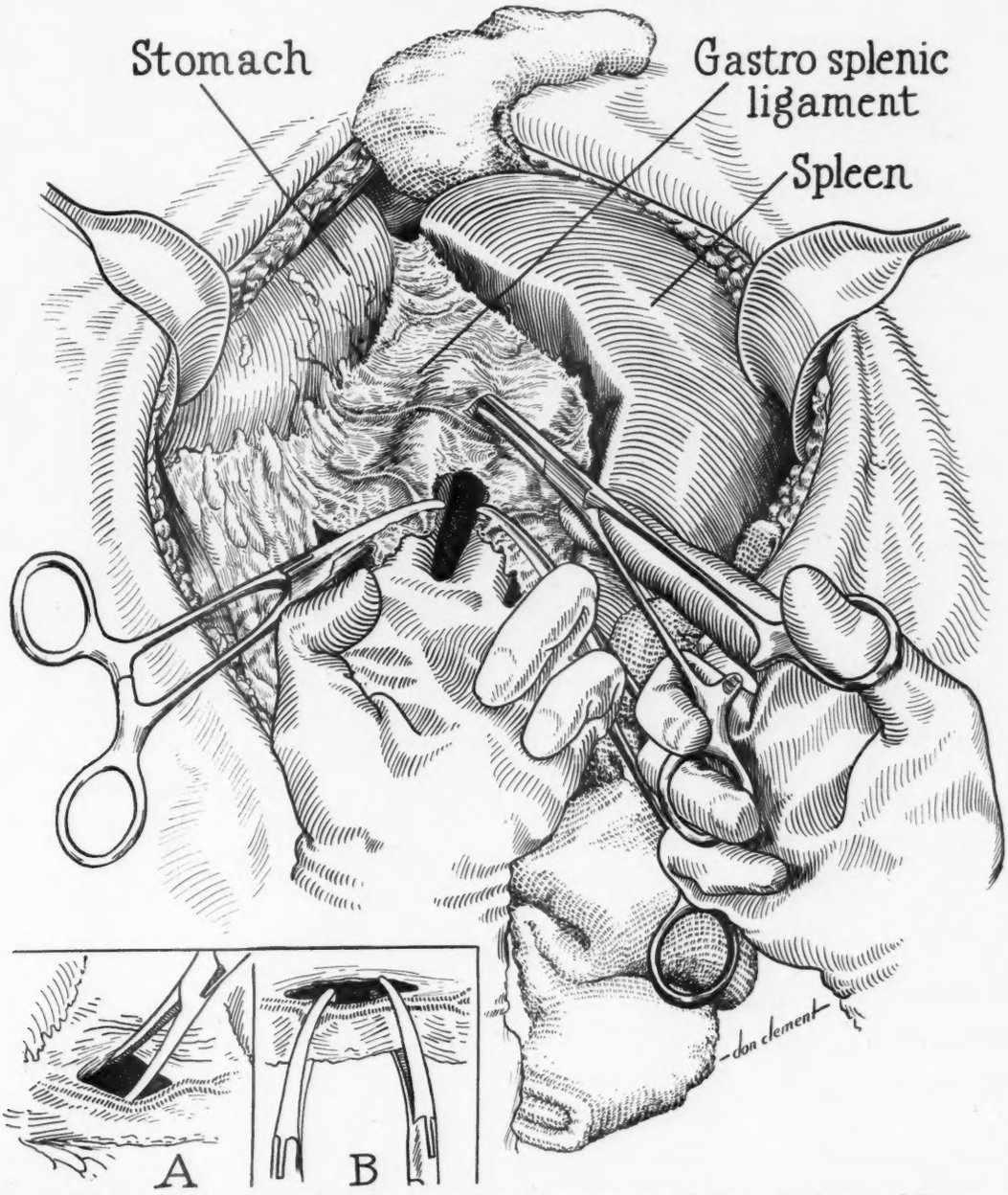

Fig. 7. Division of the gastrosplenic ligament. Two fingers separate it from the lienorenal ligament below. Division of this structure is easily accomplished in small bites of tissue which include the large vessels, as shown in the inserts. This careful dissection is necessary to avoid injury to the stomach which may lie directly against the upper pole.

accomplish. This now leaves the spleen attached only by its true pedicle, the major vessels of which have already been secured by ligature. It is now possible to divide these under direct vision with no possible danger of wounding the stomach. (Fig. 8.) Until the gastrosplenic ligament has been

After inspection of the retracted pedicle, to be certain that bleeding is controlled, the large pack is removed and the bed of the spleen and the diaphragm inspected for bleeding points. Usually, all ooze has ceased by this time, but if not it is amazing how easy it is, once the spleen has been

removed, to isolate, clamp, and tie a few individual veins in this area.

The problem of what to do if a clamp slips, the pedicle is lost, and uncontrolled bleeding is encountered, requires consideration. Actually, this should not happen if the vessels are secured individually according to the technic just described. Rarely, a badly fractured spleen may be pulled off from the pedicle, or, as in a case seen by the author, be entirely separated from the pedicle by the initiating trauma. In such cases, unless the exposure is unusually adequate the left rectus incision should be extended to the left by a T-shaped extension. (Fig. 3.) The loose fragment of spleen is removed, the remainder of the spleen delivered and the cavity firmly packed with a huge gauze pad. By pressure on this pad, bleeding will be controlled sufficiently to permit a normal circulatory state to be maintained by a continuous blood transfusion. The main splenic artery should then be isolated by exposing it where it courses above the pancreas through the gastrocolic ligament. (Fig. 9.) This approach should be

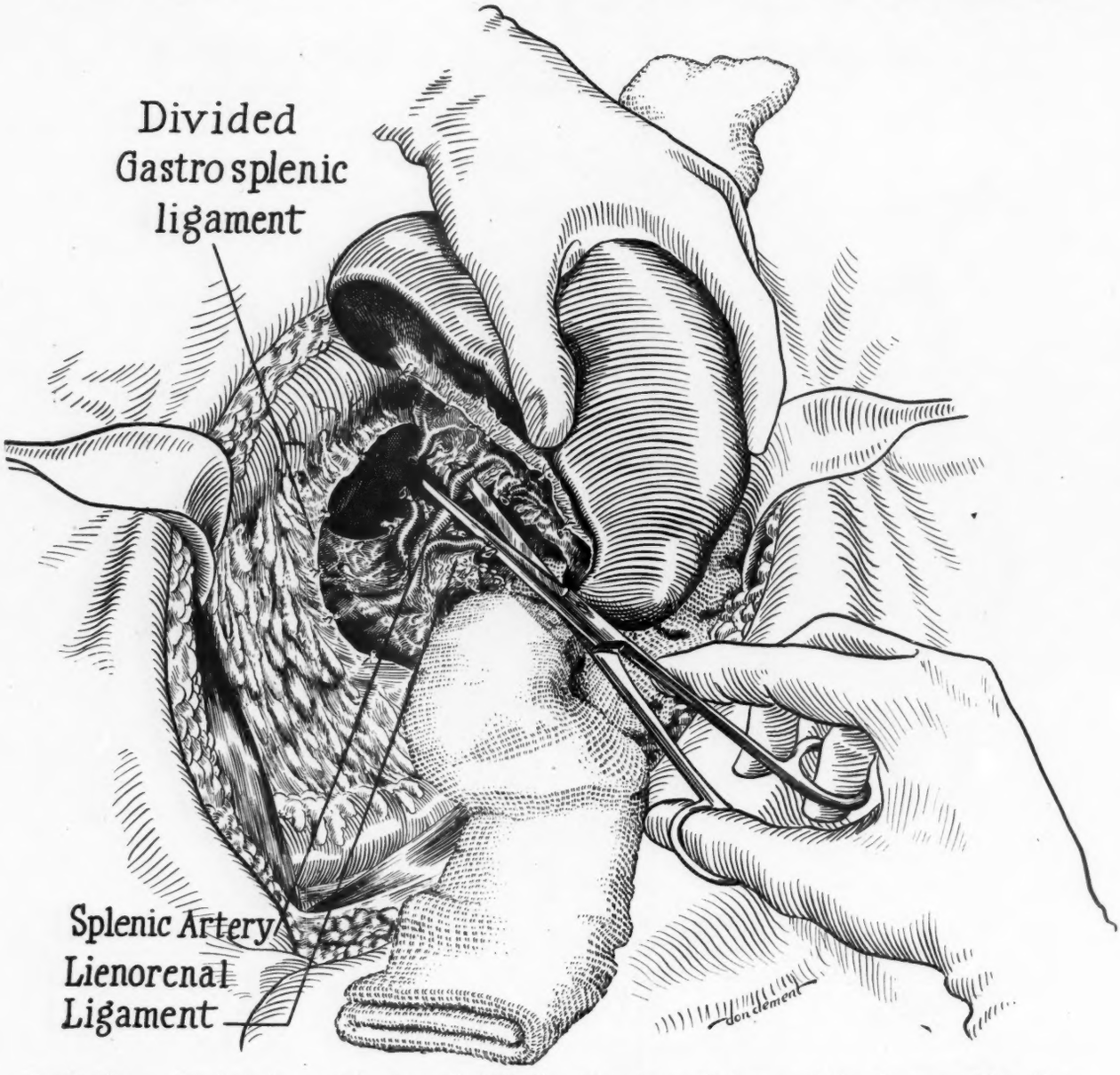

Fig. 8. The gastrosplenic ligament has been divided. The spleen is attached now only by the true pedicle (lienorenal ligament), the main artery of which has already been tied. (Fig. 6.)

made some distance away from the spleen proper, as if one were opening the lesser sac in preparing to free the greater curvature of the stomach for gastrectomy. Here one obtains easy access to the lesser sac without the troublesome bleeding, adhesions, or peritoneal reflections encountered directly over the gastrosplenic ligament. Once the lesser sac is opened, by working to the left, the tail of the pancreas and the retracted bleeding pedicle are readily identified. (Fig. 9.) Once this has been secured, the remainder of the operation can be conducted as previously described.

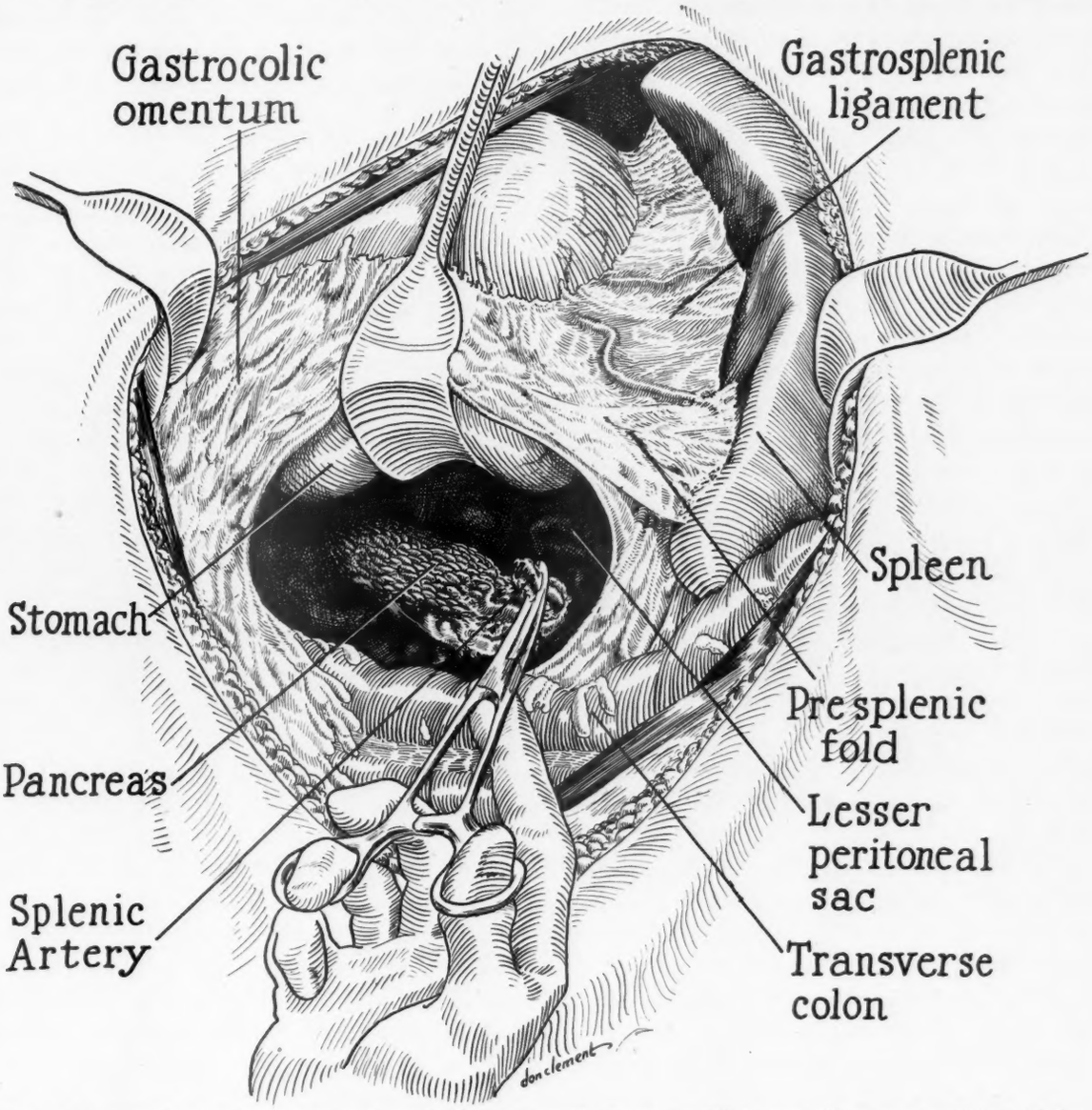

Fig. 9. Approach to splenic artery through the gastrocolic ligament. This route is most expeditious if the splenic pedicle is lost, or the spleen is torn completely from its pedicle by the initiating trauma.

At the conclusion of the operation all blood and blood clot should be removed from the peritoneal cavity. Since blood loss is so readily replaced by transfusions there is no sound basis on which to recommend deliberately leaving blood or blood clots in the peritoneal cavity. Closure should be done in layers and supported by retention sutures.

POSTOPERATIVE CARE

Shock therapy should be continued after operation until a normal circulatory state is maintained without it. In general, it is

gratifying to see how well these patients do postoperatively. Atelectasis of the left lower lobe of the lung is likely, and efforts should be directed toward its prevention. Following removal of the ruptured spleen there is normally a persistent, slight thrombocytosis and leucytosis with a relative lymphocytosis. There is no anemia and no detectable physiological abnormalities.

The development of accessory spleens scattered throughout the abdomen may occur after traumatic rupture of the spleen.[5] This phenomenon may play a compensatory rôle.

Splenectomy need not constitute a contraindication to military service.

SUMMARY

A technic of splenectomy for trauma is presented in detail. Attention is called to the fundamental contributions of Balfour to this field. Certain features of the pre- and postoperative care are discussed.

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Announcement: The American Association for the Surgery of Trauma announce that plans have been formulated for their annual meeting to be held June 26, 27, 28, 1946, at the Plaza Hotel, San Antonio, Texas.

COMPLETE DUODENAL OBSTRUCTION IN THE NEWBORN*

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COMPLETE obstruction of the duodenum in the newborn constitutes a grave surgical emergency. A prompt diagnosis combined with remedial surgery offers the only opportunity for survival.

Before corrective therapy can be effectively employed one must have a clear understanding of the manner in which various anatomic abnormalities can and do effect these troublesome occlusions. The provocative mechanisms can be conveniently grouped into those factors effecting an intrinsic occlusion and those producing extrinsic compressions of the bowel lumen.

The intrinsic occlusions represent a failure of the duodenum to form a patulous lumen. During a certain phase in its embryological development the intestines consist of a solid cord. Later, the central cells disintegrate and form large vacuoles, and as these vacuoles coalesce, the intestinal cord becomes tubular. Faulty tabualization results in a variety of congenital anomalies. If the entire duodenum remains as a solid cord, the condition is known as complete congenital atresia. Occasionally, a partial or segmental vacuolation occurs causing a portion of the duodenum to be normal while other segments have no lumen. In rare instances the entire duodenum may be patulous except for a small transverse diaphragm which effectively inhibits the flow of duodenal fluids into the jejunum.

The extrinsic obstructions are caused by external compression of a normally developed duodenum.[8] Rotation of the midgut may be accompanied by the formation of strong bands of cicatricial tissue around the descending and transverse segments of the duodenum so as to compress its lumen. Occasionally, the cecum may lie directly over the duodenum and exert sufficient external pressure to obliterate the duodenal lumen. Faulty fixation of the mesentary of the small bowel frequently obstructs the intestine at the duodenal jejunal angle.[8] All of these conditions, whether of intrinsic or extrinsic origin, do produce acute obstructions of the duodenum.

SYMPTOMS

The obstruction and the ensuing pathologic changes are well established by the time the child is born as they may occur before the fifteenth week of fetal life. Vomiting is the cardinal symptom. It begins shortly after the first feeding and becomes progressively worse until it assumes a pernicious nature. As a general rule the occlusions lie below the ampulla of Vater, hence the vomitus is stained with bile while the meconium is acholic. Should there be a complete atresia of the duodenum or if the obstruction is situated at the level of the ampulla of Vater, neither the regurgitated food nor the stools contain biliary pigments. In the suprapapillary obstructions the acholic vomitus consists of ingested foods mixed with gastric secretions while the meconium is stained with bile.

Whenever the obstructive agent interferes with the free flow of bile into the duodenum the infant becomes jaundiced. While icteric changes in the newborn usually portend no serious pathologic condition they may signify serious biliary obstructions. This is evidenced by the fact that one of our patients was treated for icteroneatorum for six days and it required an autopsy to convince the physician that

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a complete duodenal atresia was the sole cause of the jaundice.

Distention is invariably present and is always limited to the epigastric area while the lower abdomen remains flat. This significant clinical sign is produced by the failure of ingested air and gas forming bacteria to pass beyond the obstructed duodenum, so that the small intestines and the colon remain in a collapsed state. Peristaltic waves may be seen crossing the epigastrium from the left to the right side.

Dehydration combined with inability to absorb foods, vitamins, and essential electrolytes, soon lead to anemia, loss of tissue turgor, progressive inanition and mild hyperthermic reactions.

DIAGNOSIS

While careful observation and repeated examinations during the first twenty-four hours of life may suggest the diagnosis of an acute intestinal obstruction, it requires roentgenologic studies for accurate localization of the occlusion. Simple scout films of the abdomen may demonstrate a gaseous distention of the stomach and duodenum while failing to outline gas bubbles in the jejunum, ileum or colon. (Fig. 3.) One should always inquire whether an enema has been given prior to the *x*-ray examination, for if air has been introduced into the colon it cannot be differentiated from ingested air and may lead to an erroneous conclusion of a patent intestinal tract. Films should be taken in an anteroposterior, anterolateral positions and with the child in the inverted and upright postures. If the gas bubbles do not change their position, a fixed congenital deformity can be assumed. Neuhauser[7] found that the obstructed duodenum may rupture during neonatal life thus liberating the sterile meconium into the peritoneal cavity. He found that in such instances the meconium contains depositions of calcium which are readily detected by roentgenographic studies. Such deposits were confined to the proximal segment of obstructed gut and to the intraperitoneal areas containing the meconial c'umps.

When contrast media are used for *x*-ray studies, care must be exercised to prevent the aspiration of this material, as the swallowing, coughing and expectorating reflexes of these frail infants are very immature. This hazard can be greatly minimized by injecting the barium mixtures through a small Levine tube which has been previously passed into the gastric lumen. The barium clearly delineates the dilated stomach and megladuodenum thereby effectively localizing the obstruction. A differential diagnosis between complete obstruction, and hypertrophic pyloric stenosis can be readily made by progress films, for in the latter a small amount of barium invariably passes into the jejunum. After completion of the *x*-ray studies it has been our practice to aspirate the barium mixture by gently lavaging the stomach. This not only minimizes the dangers of aspiration pneumonia but facilitates respiration by relieving the gastric distention. Detection of a faulty rotation of the midgut can be made by employing a barium enema. The abnormal position of the cecum is diagnostic. Such studies should never be attempted unless the stomach and duodenum have been decompressed as the resulting colonic distention might seriously embarrass respirations. Ladd[4] affirms that the information acquired from the roentgenologic studies of the colon is not worth the risk it entails as the operative exposure is the same for all types of duodenal obstructions.

TREATMENT

It is imperative that these frail infants be thoroughly prepared for the traumatic insults of surgery. Operations which were formerly prohibitive can now be executed with comparative safety because of this physiological rehabilitation. It is essential that the fluid and electrolytic balance be changed from the negative to a positive phase, taking care not to overburden the immature circulatory and renal systems.

Ladd and Gross[4] advise that all replacement fluids should be given in small amounts and at regular intervals. The thighs, buttocks and flanks afford excellent receptacles for non-irritating fluids. In the presence of extreme dehydration solutions of 5 per cent glucose containing cevitamic acid, vitamin B complex, vitamin K and essential ammino acids are administered by the intravenous route.

Transfusions of whole blood are invaluable in combatting the existing anemia and reinforcing the babies inadequate immunological reactions. It is well to remember that a hemoconcentration due to a dehydration can give a normal blood count when in reality a serious anemia exists.

Constant decompression of the dilated stomach can be readily accomplished by using the indwelling Levine tube. This permits the atonic gastric and duodenal walls to regain their normal tone thus expediting subsequent surgical correction.

Many of the postoperative pulmonary complications come from the unwise selection of the anesthetic agent. Ether irritates the pulmonary tree thereby producing thick tenacious mucus which these babies are unable to cough up and expectorate, thus predisposing to atelectasis. Theoretically, the infiltration of 1 per cent solution of novocaine would be ideal but we have found that it does not provide adequate muscular relaxation for careful exploration and the added trauma necessary to prevent eviscerations adds to the operative shock. Brown[1] maintains that these tiny infants do not tolerate too much novocaine and he reports examples of lethal poisoning from overdosage. Our anesthetist, Dr. Scott Smith,* believes that cyclopropane is a safe and useful agent. During the past months he has used cyclopropane in eight infants, varying from fifteen hours to two weeks of age, who have undergone operations for hypertrophic pyloric stenosis or duodenal obstructions. Whenever complete muscular relaxation has been essential he has supplemented cyclopropane anesthesia by administering curare (intocostrin-Squibb) with the most pleasing results. In every instance the babies were awake at the termination of the operation and all reflexes were active. Such agents, however, must be administered by an expert.

The primary objective of every operation is to relieve the obstruction in the quickest and safest manner possible.[6] Unfortunately, procedures which are applicable to adults, such as enterostomies and Mikulicz decompressions, cannot be used in infants because of the profound physiological disturbances which they produce. Peritonitis is common with these operations as the immunoilogical defenses are not fully developed. Reconstructive measures should aim to divert the essential digestive ferments into those segments of the intestinal tract where nature intends them to function. The more closely this can be realized the less severe will be the postoperative digestive dysfunctions.

When confronted with a complete atresia of the duodenum one must relieve the gastric obstruction and decompress the obstructed extrahepatic bilary radicals. This requires two separate procedures. We have employed both the antecolic and the posterior gastrojejunostomies in relieving the duodenal stasis. The antecolic type, as popularized by Lahey,[5] can be executed more rapidly and with less trauma than any other variety and should be used if the infant is in a precarious state. The biliary stasis can best be corrected by performing a cholecystogastrostomy.[2] Fortunately, the distended gallbladder and the dilated stomach are contiguous so no difficulty is encountered in forming a diversional biliary stoma. In such instances we always pull the tip of the indwelling Levine tube through the newly formed cholecystogastric stoma so that it lies within the lumen of the gallbladder.[3] Constant suction keeps the gallbladder decompressed by syphoning off the static bile, thus expediting hepatic recovery. It also diminishes the intracystic

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pressure of the gallbladder thus reducing the tension of the stomal suture lines and minimizes the dangers of bile leakage with its resulting peritonitis.

Occasionally, acute obstructions are caused by compressive cicatrical bands which cross the lower end of the descending duodenum or the proximal portion of the transverse segment. In many instances one needs but to divide the restraining bands of scar tissue to establish the patency of the duodenum. If the contents of the proximal duodenum can be manually forced across the obstructed zone no additional surgery is required. We have found, however, that the involved segment may have a concomittant atresia and unless this can be corrected some short circuiting procedure such as duodenojejunostomy or gastrojejunostomy must be employed to relieve the obstruction. All dissections must be done under direct vision so as not to injure the blood supply of the duodenum. The biliary tract is not involved hence requires no attention.

Non-rotation or incomplete rotation of the midgut may produce so much tension on the short superior mesenteric vessels that they effect an acute obstructive angulation of the duodenum at its junction with the jejunum. The superior mesenteric artery may lie in front of or behind the duodenum and be surrounded by obscuring bands of dense adhesions. In some instances releasing the adhesions may correct the kinking but as a rule the vascular pedicle is so short that it still exerts enough pressure to occlude the intestinal lumen. It is obvious that the mesenteric pedicle cannot be elongated so a duodenojejunostomy should be employed. On one occasion we encountered such a situation and had no difficulty in performing the duodenojejunostomy but further exploration revealed that an incomplete rotation of the midgut had caused a partial volvulus so that it required the execution of a Wangensteen operation for non-rotation.[9] These are long, tedious operations and are not well borne by the infant.

The disparity in size between the collapsed segment and the distended loops presents a tantalizing problem in anastomosis. The distal loop appears as a small cord of tissue for the lumen has never been distended by traversing fluids. Wangensteen[9] identifies the lumen by injecting mineral oil into it and manually forcing the lubricating mixture along the collapsed bowel. This not only determines the patency of the gut but distends its walls so that anastomotic procedures can be easily executed. We attempted this technic but were unable to tell when the point of the exploring needle had entered the lumen of the bowel and the injected oil merely extravasated along its muscular coats. It was necessary to open the jejunum and introduce the oil directly into the lumen by means of a small urethral catheter.

When the anastomosis is completed the indwelling Levine tube is pulled through the newly formed stoma so that its tip lies in the distal loop of collapsed bowel. A slow continuous drip of 5 per cent glucose containing all the essential vitamins furnishes adequate nourishment and fluids. If these are well tolerated periodic feedings of a suitable formula or breast milk can be given. Should the bowel become distended continuous decompression soon relieves the annoying distention.

CASE REPORTS

Case 1. *Occlusion of the Duodenum by Extrinsic Adhesions:* B. S. S., a full term baby having a birth weight of seven pounds fifteen ounces vomited its first feeding and every one thereafter. The skin was dry and the turgor of the tissues was poor. We saw her on the fourth postnatal day, at which time she weighed but five pounds six ounces. No attendant, either in the delivery room or in the nursery had observed the passage of flatus or meconium. The epigastrium was distended and tympanitic but no peristaltic waves were visible. A scout film revealed an enormous dilation of the stomach and first portion of the duodenum but no gas was visible in the small intestines or colon. A dilute solution of barium was introduced into

the stomach through a Levine tube but none of the barium passed beyond the transverse duodenum. (Fig. 1.) The barium was immediately aspirated from the stomach by means of gentle irrigating lavages. A mild flushing of the colon recovered mucoid material devoid of bile pigments, milk curds or squamous epithelial cells. The colon occupied its normal position. It was assumed that we were dealing with an infra-ampullary duodenal obstruction.

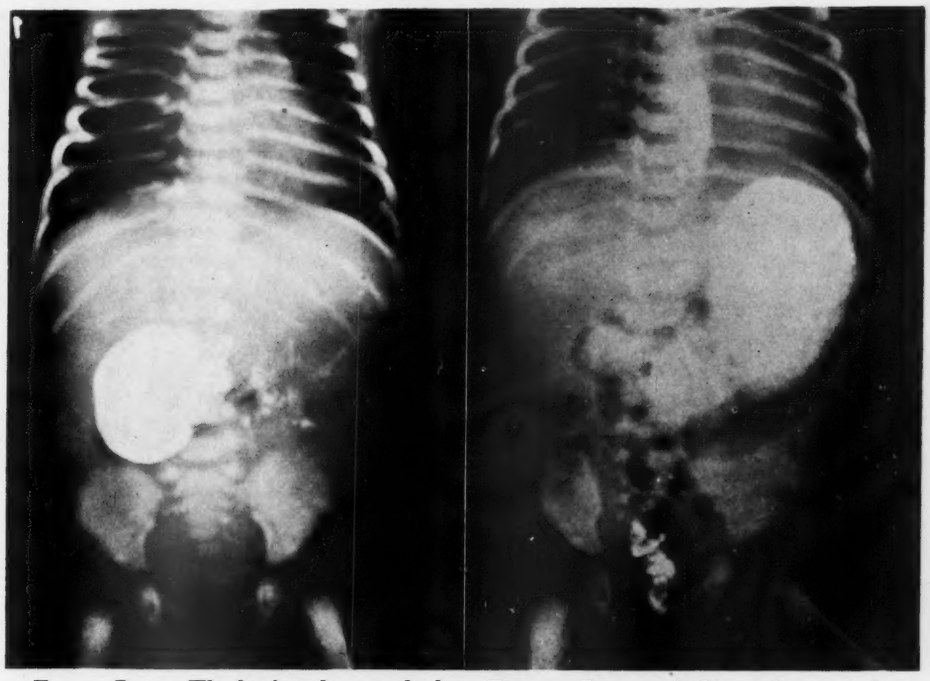

Fig. 1. Case 1. The barium has reached the obstructed duodenum. Note the degree of dilatation. No gas bubbles are visible in the small intestines or colon, thus confirming the diagnosis of congenital atresia of the duodenum.

Fig. 2. Case 1. A film taken on the fourth postoperative day, thirty minutes after the ingestion of the barium. The degree of duodenal dilatation has diminished. Barium can be seen passing through the newly formed stoma into the jejunum. The rectosigmoid contains barium which had been ingested on the previous day.

The preoperative regime consisted of correcting the dehydration, replenishing the electrolytes, and combatting the existing anemia by repeated transfusions of whole blood. The abdomen was opened under cyclopropane anesthesia. The dilated stomach was further decompressed by continuous suction on the indwelling Levine tube. A dense band of adhesions arising from the root of the mesentery were seen to cross the transverse segment of the duodenum to become attached to the posterolateral parietes. These adhesions were divided thereby liberating the duodenum. However, none of the contents of the upper duodenum could be expressed beyond the point of obstruction. Whether the prolonged pressure of the extrinsic cicatricial bands had held the walls of duodenum in such firm apposition that they had fused, or whether the obstruction presented a regional atresia could not be determined. The collapsed jejunum was opened and found to have a normal lumen as was evidenced by the ease with which 10 cc. of mineral oil could be forced along its lumen. A posterior gastrojejunostomy was accomplished by the usual technic. The indwelling Levine tube was pulled through this stoma so that its tip was placed in the lower jejunal loop. Three hours later feedings of breast milk were given through the tube and pleasing convalescence resulted. Fluoroscopic studies with barium demonstrated a normal functioning gastroenterostomy. (Fig. 2.) This youngster is now one year of age and in the best of health.

Case II. *Duodenal Obstruction from Faulty Rotation of the Cecum:* B. J., female baby, full term, weighed six pounds ten ounces. The obstetrician commented that the distended

epigastrium made the delivery very difficult. She vomited in the delivery room and had progressive regurgitation with every feeding.

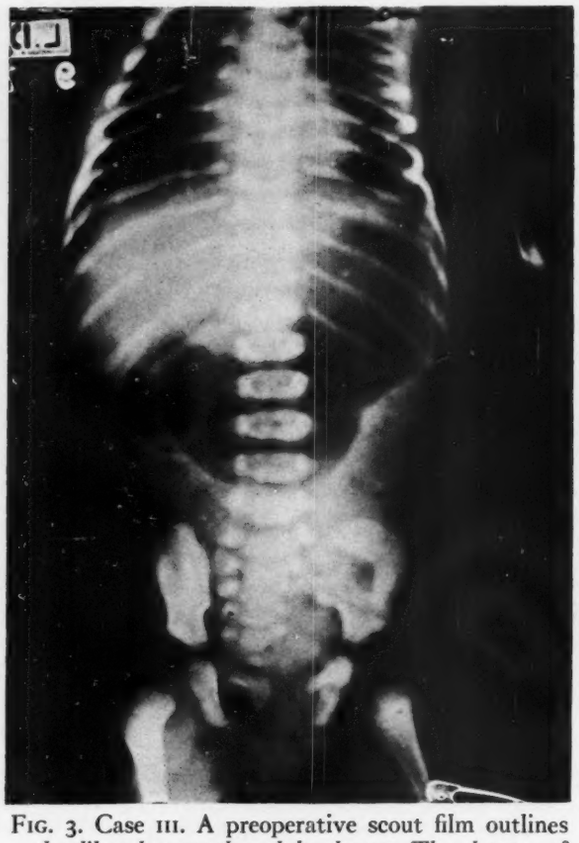

Fig. 3. Case III. A preoperative scout film outlines the dilated stomach and duodenum. The absence of gas in the small intestines and colon is pathognomonic of congenital obstructions of the duodenum.

The vomitus contained bile. Antispasmodic drugs, special formulas, and blood transfusions all failed to halt the dehydration, emaciation and rapid loss of weight.

We saw the baby on its sixth day of life, at which time it was in a very precarious condition. After careful preoperative preparation the abdomen was opened under cyclopropane anesthesia. The stomach and duodenum seemed to fill the entire abdominal cavity. The cecum lay just below the distal end of the stomach and from it a dense band of reflected peritoneum passed over the descending duodenum thus effectively compressing its lumen. After releasing the abnormal adhesions the duodenal canal refused to be dilated. Pressure on the dilated stomach did not force the fluids beyond the point of obstruction. The baby was in such a precarious state that an antecolic gastrojejunostomy seemed advisable. After opening the collapsed jejunal lumen and forcing mineral oil along its canal the anastomosis was performed and the Levine tube inserted into the distal jejunal loop. No attempt was made to correct the malrotation. In spite of the most zealous postoperative therapy this emaciated youngster died on the fourth postoperative day from a bronchopneumonia.

Autopsy studies demonstrated a functioning stoma and a complete occlusion of the duodenal wall at the point where the adhesions had been divided. An early diagnosis with prompt surgical correction might have saved this baby.

Case III. *Congenital Atresia of Duodenum:* J. C., a female, full term, weighed five pounds and ten ounces at birth. The obstetrician noticed that the epigastrium was greatly distended and the skin had deep icteric tinge. A pernicious and persistent vomiting followed the first feeding. The acholic vomitus consisted of gastric juices and milk curds. No meconium was passed even though given small colonic flushing and the returning fluids were acholic. In spite of daily transfusions of 30 cc. of whole blood a progressive anemia developed.

The attending physician made the presumptive diagnosis of icterus neonatorum.

The baby was six days old when first seen in consultation. The weight was four pounds ten ounces. The icteric index was 85, the urine contained 4 plus bile but lavage of both the stomach and bowel were negative for bile pigments. Scout films revealed a dilation of the stomach and duodenum while the ileum was not visualized, but the colon contained a few pockets of gas. (Fig. 3.) The colonic gas was thought to have been introduced when giving the enema. A diagnosis of complete atresia of the duodenum with obstruction of the bile ducts was made, but as the baby was moribund operative intervention was deferred. She died two hours later.

Autopsy findings demonstrated the duodenum to consist of a solid cord, having no lumen. The gallbladder and bile ducts were enormously dilated as the atresia had effectively obstructed the ampulla of Vater. The enlarged liver was deeply stained with bile pigments resembling an adult type of biliary cirrhosis.

In spite of these extensive malformations such conditions are amenable to surgery. Had an early diagnosis been made and the proper preoperative treatment

administered the defects could have been remedied by doing a gastrojejunostomy and cholecystoenterostomy. It is interesting how well the fetus tolerates these congenital deformities and it is not until birth that toxic symptoms develop. Hicken and Crellin[2] reported five cases of congenital atresia of the bile ducts and outlined remedial procedures. It is imperative that the provocative cause of the jaundice be accurately determined if these patients are to be saved.

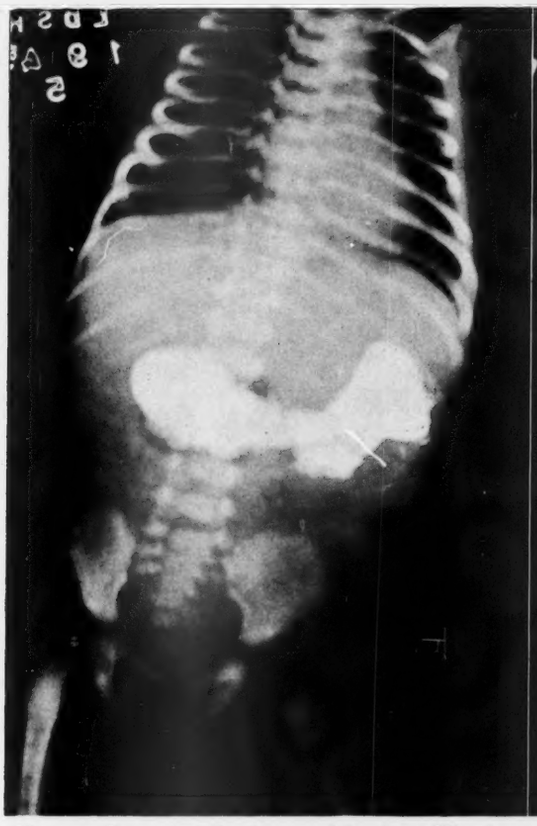

Fig. 4. Case iv. The ingested barium fills the dilated stomach and duodenum but fails to pass into the jejunum. The absence of gas in all segments of the lower intestinal tract suggests the presence of a congenital obstruction of the duodenum.

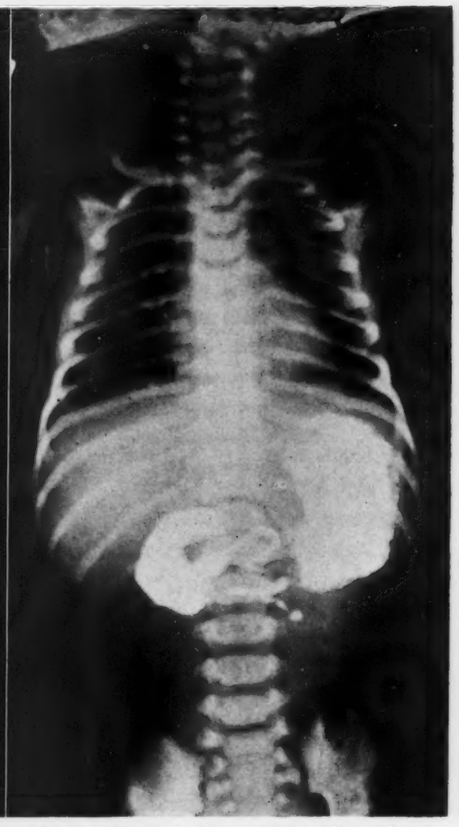

Fig. 5. Case iv. A progress film taken on the third postoperative day shows the barium escaping through the stoma into the upper jejunum. The entire intestinal tract is now patent as attested by the presence of gas in various segments of the intestinal tract.

Case iv. *Transverse Septal Diaphragm of the Duodenum:* B. M., a female, weighing six pounds four ounces, appeared normal at birth. On the second day a peculiar cyanosis appeared which was readily relieved by oxygen therapy, but no pulmonary or cardiac abnormalities could be demonstrated. There was a spontaneous evacuation of bile stained meconium. A persistent and troublesome vomiting contributed to a rapid dehydration and emaciation. A barium meal revealed a complete obstruction of the first part of the duodenum as none of the barium was seen to pass into the jejunum. (Fig. 4.) The small intestines and colon were devoid of gas bubbles. The extremities were slightly spastic. A presumptive diagnosis of a complete atresia of the supra-ampullary portion of the duodenum and cerebral birth injuries seemed reasonable.

Dr. Scott Smith administered cyclopropane anesthesia supplemented by the intravenous injection of intocostrin (curare-Squibb). The stomach, duodenum, and gallbladder were enmeshed in a dense mass of adhesions, which were thought to have been produced by a neonatal pericholecystitis. After liberating these structures a heavy strand of adhesions was seen to cross the transverse duodenum.

This cicatricial band was divided and attempts were made manually to force the gastric contents along the duodenum, but an impassable obstruction was encountered just above the ampulla of Vater. The anterior surface of the stomach was opened and a small catheter was passed into the duodenum but the persisting obstruction was again encountered. As the patient's condition was precarious an anterior gastrojejunostomy was performed and a Levine tube was threaded through the newly formed stoma so that it lay in the distal loop of the jejunum.

During the first three days the postoperative course was very pleasing as all jejunal feedings were retained. (Fig. 5.) The following morning she developed a fulminating hyperthermia, an untractable cyanosis, and a progressive pulmonary congestion which terminated fatally six hours later. An autopsy revealed a bilateral apoplexy of extensive proportions, small petechial hemorrhages through the brain, diffuse pneumonitis and a localized peritonitis. Apparently the intracranial hemorrhages accounted for the preoperative cyanosis and the hemorrhagic changes of the adrenal glands produced the hyperthermia. It seems reasonable to assume that these complications resulted from birth injuries. The gastrojejunostomy stoma was functioning, for stomach contents were found in the ileum. (Fig. 5.) There was slight leakage at the stoma for small amounts of the gastric juices had seeped out between the sutures. Interestingly, both the abdominal wound and the gastric stoma presented no evidence of healing.

Case v. *Complete Atresia of Duodenum and Bile Ducts:* L. C., a female, had suffered from a pernicious vomiting during the first three days of life. Biluria, acholic stools, intense jaundice, malnutrition, melena and enlargement of the liver suggested the diagnosis of atresia of the duodenum and bile duct. In spite of an energetic attempt to prepare her for operation she died the following day. Postmortem studies demonstrated a complete atresia of the gallbladder, bile ducts and duodenum. Such extensive abnormalities cannot be corrected.[2]

COMMENTS

Acute obstructions of the duodenum in the newborn must be classed as surgical emergencies. Delay merely invites a prohibitive mortality as indicated in this study. In our series of five cases there was an overall mortality of 80 per cent. One patient was moribund when surgical consultation was obtained so no operation was attempted. Two babies had remedial conditions but were so desperately ill that they were unable to withstand the operative shock. One infant had such an extensive atresia of the biliary radicals that correction was impossible. Only one infant made a complete recovery. In retrospect, however, we sincerely believe that an early diagnosis combined with corrective surgery might have saved four of these five patients. Three unnecessary deaths seem to be a high price to pay for procrastination.

Case	Anatomy	Operation	Result
S. S. Female	Compressive adhesions transverse duodenum Atresia duodenum	Anterior gastrojejunostomy	Recovery—well 1 year
B. J. Female	Atresia transverse duodenum Faulty rotation of cecum	Anterior gastrojejunostomy	Died—pneumonia 4th postoperative day
J. C. Female	Atresia duodenum at ampulla Vater	No operation; moribund when seen	Confirmed by autopsy
B. M. Female	Transverse duodenal diaphragm, supra-ampullary	Anterior gastrojejunostomy	Adrenal apoplexy; died 5th postoperative day; cerebral hemorrhage
L. C. Female	Complete atresia of duodenum and bile ducts	Exploration only	Died 5th postoperative day

This study emphasizes one other important fact, namely, that the surgeon cannot assume that the cicatricial bands which are compressing the duodenum are solely responsible for the obstruction. Occasionally, as in two of our patients, there may be an associated segmental atresia of the duodenal lumen. Unless both conditions are corrected the obstructive syndrome continues.

SUMMARY

1. Five cases of acute obstruction of the duodenum occurring in the newborn are presented.

2. A discussion of the various anatomic abnormalities encountered, symptomatology, roentgenograph diagnosis and methods of treatment are presented.

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Announcement: The American Association for the Study of Goiter announce that their annual meeting will be held at the Drake Hotel, Chicago, Illinois, June 20, 21, 22, 1946. This will be the first meeting held since the beginning of the War.

NON-ECLAMPTIC LATE TOXEMIAS TREATED BY VITAMIN E

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FOR several years past[1,2] a study has been made of the effect of administering vitamin E to women suffering from the non-eclamptic late toxemias of pregnancy.

What follows is an analysis of 128 consecutive, unselected "toxemias" of this type seen in private practice and so treated with vitamin E. Many of these patients had salt restriction as well, but no other therapy was used, and all were ambulant throughout, except Case 94. Many of them came under care in the first trimester of pregnancy, as will appear. Large doses of vitamin E were often required, especially toward term.

The late toxemias were regarded for the purpose of this study as the state of those patients displaying edema and/or hypertension and/or albuminuria in the latter half of pregnancy. These toxemias were held to be non-eclamptic if the blood estrogen level was high or "positive."[3]

The writer has repeatedly demonstrated,[4,5] as have others,[6] that a low estrogen value is a fundamental feature of either true pre-eclampsia or convulsive eclampsia. Indeed, clinical differentiation between the late toxemias is generally impossible without blood estrogen assays.

Such differentiation is essential if vitamin E is to be used therapeutically for pregnancy toxemias, for E probably is harmful to pre-eclamptics.[5] Surely endocrine therapy should always be preceded by endocrine diagnosis, and vitamin E is a powerful anti-estrogen.[7]

It is obvious that the designation of advanced pregnancies as toxemic when presenting any or all of the triad of albuminuria, edema or hypertension leaves much to be desired. Those who favor the nomenclature of the American Committee on Maternal Welfare must look at it askance. But this ancient classification is simple, certainly includes all the cases which should be considered, and is the one generally used by practitioners and most specialists. Of course it includes, unfortunately for scientific accuracy, all those patients having renal-vascular damage long antecedent to pregnancy. But these last could not react very favorably to any therapeutic measure used only during a pregnancy; therefore, the clinical results that were achieved should not be biased in its favor. Our scales have thus been weighted against E deliberately.

DATA AND ITS ANALYSIS

The clinical data on the 128 women are listed in Table I. The blood pressures of the 128 women are as set forth in Table II.

Most of these women came under observation in the first trimester of pregnancy, as a glance at Table I will show. However, a large percentage, 79 per cent to be exact, did not attain a systolic of more than 160 or a diastolic of more than 100 during what remained of their pregnancies. Moreover, at the time of delivery, fully 43 per cent of the women with elevated pressures had pressures appreciably lower than the highest they had exhibited during gestation.

It is to be remembered that only one of these women was put to bed. The remainder did their housework as usual.

The beneficial effect of E therapy is demonstrated even better by observations made on their edema. Thirty-eight per cent of the women had no edema at delivery, 30 per cent had only slight edema, and but 32 per cent revealed more than that.

The figures on albuminuria are also of

TABLE I

Case	Under My Care (in Weeks)	When First Seen (in Weeks)	First Sign of Toxaemia and When (in Weeks)	Highest Blood Pressure and When (in Weeks)	Blood Pressure at Time of Delivery	Oedema at Delivery	Albumin at Delivery	Stage at Delivery (Wks.)	Manner of Labour and Delivery	Child	Later Course	Remarks
1	18	22	Hypertension @ 32	174/116 @ 38	140/98	0	tr.	40	Spont.	One forearm absent. 7½ lb. boy.	?	
2	20	20	Hypertension before seen.	198/104 @ 28	188/102	+	0	40	Spont.	Stillborn 14½ lb. male. Erythroblastosis.	196/112 2 yrs. later.	
3	28	12	Oedema @ 37	134/76 @ 38	126/76	+	0	40	Spont.	8′5″ boy.	Normal 2 yrs. later.	
4	30	10	Hypertension and oedema before seen.	150/100 @ 37	138/88	0	0	39	C. S.	7′8″ girl	Sterilized. 170/120 3 yrs. later.	
5	35	5	Hypertension before seen.	166/98	154/94	++	0	39	Med. ind.	8′5″ boy	9 wks. later 156/98.	
6	34	6	Oedema @ 25	142/94 @ 40	142/84	+++	0	40	Spont.	8′4″ girl	Slight oedema 3 yrs. later.	
7	18	22	Hypertension @ 36	146/86 @ 39	108/76	0	0	40	Spont.	8′5″ boy.		
8	31	9	Oedema @ 33	132/80 @ 34	128/70	++++	0	40	Spont.	7′9″ girl.	Normal for 2 yrs.	
9	32	8	Oedema @ 37	138/94 @ 38	134/84	++	0	40	Spont.	7′4″ boy—idiot.	Normal 5 yrs. later.	
10	28	12	Hypertension @ 32	144/96 @ 37	134/94	+	0	39	Med. ind.	8′8″ boy.	Normal 3 yrs. later.	
11	19	21	Hypertension before seen.	238/138 @ 38	170/92	0	3+	38	C. S.	8′14″ girl	Diabetis 5 yrs. later.	Diabetes
12	35	5	Hypertension @ 11	160/96 @ 11	136/96	0	0	40	Spont.	7′ girl		
13	18	22	Albumin @ 31	140/100 @ 37	136/100	0	0	40	Spont.	7′8″ boy.	Normal 14 mos. later.	
14	36	4	Hypertension @ 37	144/104 @ 39	144/104	+	0	40	Spont.	7′ girl		
15	26	14	Hypertension @ 32	140/94 32	132/82	+	0	40	Spont.	7′ girl.		
16	32	8	Hypertension before seen.	164/80 @ 30	150/70	0	0	40	Spont.	7′14″ boy.		
17	24	16	Hypertension @ 30	164/88 @ 38	148/76	0	0	40	Spont.	6′6″ girl		
18	36	4	Oedema @ 36	138/98 @ 40	116/56	3+	0	40	Spont.	8′ boy		
19	35	5	Oedema & albumin @ 27	154/104 @ 40	154/104	+	0	40	Spont.	8′2″ girl	Normal 5 yrs. later.	
20	20	20	Hypertension @ 30	146/94 @ 40	144/90	0	0	40	Spont.	8′ boy	Normal 1 yr. later	
21	31	9	Oedema @ 26	150/82 @ 42	150/80	+	0	42	Med. ind.	9′ boy	Glaucoma 6 yrs. later.	
22	18	22	Albumin & hypertension @ 30	146/116 @ 35	140/116	0	4+	33	Spont.	Stillborn breech	?	Pyelitis after 30th wk. Bleeding daily from 25–29 wks. Praevia suspected but excluded.
23	34	6	Hypertension @ 17	140/94 @ 36	140/94	+	0	36	Spont.	6′ girl		
24	34	6	Hypertension @ 27	194/104 @ 40	194/104	0	?	40	Med. ind.	6′9″ girl	.....	Off vit. E for 3 dys. before delivery. B.P. jumped then from 136/94 to 194/104 in last 12 dys.

TABLE I (*Continued*)

Case	Under My Care (in Weeks)	When First Seen (in Weeks)	First Sign of Toxaemia and When (in Weeks)	Highest Blood Pressure and When (in Weeks)	Blood Pressure at Time of Delivery	Oedema at Delivery	Albumin at Delivery	Stage at Delivery (Wks.)	Manner of Labour and Delivery	Child	Later Course	Remarks
25	18	22	Hypertension @ 29	148/104 @ 38	134/100	0	0	41	Spont.	7′7″ girl	.....	Temporary amblyopia in last 3 wks. B.P. 144/92 four mos. p.p.
26	13	17	Hypertension @ 30	142/94 @ 35	140/94	+	0	39	Spont.	6′2″ girl	.....	Appendectomy for abruptio placentae (!) @ 3 mos. Pain remained until controlled by E. Idiosyncrasy to E developed later.
27	20	20	Oedema @ 27	150/88 @ 38	124/84	4+	0	40	Med. ind.	8′2″ girl	.....	Hydatid mole 8 mos. later.
28	15	25	Hypertension @ 36	144/104 @ 38	120/88	+	0	41	Spont.	7′4″ boy		
29	13	17	Hypertension @ 18	152/104 @ 37	142/84	0	0	40	Spont.	8′1″ girl		
30	12	18	Oedema @ 20	132/94 @ 34	132/94	2+	0	37	Med. ind.	7′4″ girl		
31	20	20	Hypertension @ 28	162/118 @ 34	164/116	2+	0	34	Med. ind.	Craniospinorachischisis.	140/96wk. after delivery.	
32	26	14	Oedema @ 38	134/98 @ 40	134/98	4+	0	40	Med. ind.	8′14″ girl		
33	15	25	Oedema @ 36	146/100 @ 36	144/94	2+	0	36	Spont.	6′ boy		
34	31	9	Hypertension @ 22	150/54 @ 30	132/70	4+	0	39	Spont.	7′6″ girl		
35	16	24	Hypertension before seen.	146/94 @ 36	140/80	0	0	38	Spont.	7′12″ girl		
36	29	11	Hypertension @ 34	134/96 @ 39	134/96	0	0	40	Spont.	8′ girl		
37	28	12	Hypertension @ 16	154/94 @ 41	154/94	3+	0	41	Med. ind.	7′9″ boy		
38	25	15	Hypertension @ 18	150/88 @ 37	128/82	2+	0	40	Spont.	9′8″ boy	1½ yrs. later B.P. 144/88	
39	26	14	Oedema @ 31	118/84 @ 37	114/76	3+	0	38	Spont.	8′ boy		
40	24	12	Hypertension before seen.	152/84 @ 21	116/76	+	0	36	Spont.	9′13″ boy		
41	28	2	Hypertension before seen	154/96 @ 17	110/82	0	0	30	Spont.	anencephalic with clubfeet.	5 mos. later B.P. 138/70	
42	24	14	Oedema @ 21	140/100 @ 38	140/100	0	0	38	Med. ind.	8′1″ boy	9 mos. later 120/70	
43	12	28	Oedema @ 36	110/72 @ 40	110/72	2+	0	40	Spont.	6′1″ girl		
44	26	12	Oedema @ 31	136/90 @ 38	136/90	3+	0	38	Spont.	7′ boy		
45	24	16	Hypertension @ 25	140/120 @ 28	128/86	0	0	40	Spont.	7′ boy		
46	29	11	Oedema @ 23	134/84 @ 40	134/84	3+	0	40	Med. ind.	7′2″ girl		
47	25	15	Hypertension before seen	164/100 @ 40	164/100	2+	+	40	C. S.	6′ girl		Mother was an achondroplasic dwarf.
48	26	14	Hypertension @ 28	148/106 @ 39	140/100	0	0	40	Med. ind.	7′10″ girl	7 wks. later B.P. 130/0	
49	26	12	Hypertension & oedema @ 30	150/102 @ 38	150/102	0	0	38	Med. ind.	6′ girl	6 wks. later B.P. 136/88	
50	14	26	Hypertension when seen.	140/96 @ 38	136/100	0	2+	40	Spont.	7′8″ boy	14 mos. later B.P. 118/76	

TABLE I (*Continued*)

Case	Under My Care (in Weeks)	When First Seen (in Weeks)	First Sign of Toxaemia and When (in Weeks)	Highest Blood Pressure and When (in Weeks)	Blood Pressure at Time of Delivery	Oedema at Delivery	Albumin at Delivery	Stage at Delivery (Wks.)	Manner of Labour and Delivery	Child	Later Course	Remarks
51	10	26	Hypertension when seen.	156/100 @ 34	156/100	0	0	36	Spont.	mac. term girl	?	
52	30	4	Hypertension @ 9 wks.	154/106 @ 33	150/104	+	4+	34	Spont.	mac. boy	6 wks. later B.P. 140/80	
53	33	9	Oedema @ 21	120/74 @ 40	110/80	3+	0	42	Spont.	7′14″ boy	9 mos. later sl. oedema.	
54	34	6	Oedema @ 34	148/88 @ 39	148/88	+	0	40	Spont.	6′14″ boy	?	
55	26	14	Hypertension before seen.	144/90 @ 38	122/80	0	0	40	Spont.	8′4″ girl	B.P. 8 mos. later 142/102	
56	35	3	Hypertension @ 32	148/106 @ 36	138/110	0	0	38	Med. ind.	6′14″ girl	11 mos. later B.P. 140/84	Much pyelitis in pregnancy.
57	29	9	Hypertension @ 31	140/90 @ 34	130/80	0	0	38	Spont.	6′3″ girl	1 yr. later B.P. 124/74	
58	35	6	Hypertension @ 32	142/90 @ 35	142/86	2+	0	41	Med. ind.	8′4″ boy	?	
59	10	28	Oedema @ 25	178/114 @ 37	156/108	+	0	38	Spont.	9′3″ boy	3 mos. later B.P. 136/90	
60	35	5	Oedema @ 26	130/80 @ 38	130/76	4+	0	40	Spont.	9′2″ boy	?	
61	25	14	Hypertension for years.	146/104 @ 37	148/100	2+	0	39	Med. ind.	6′7″ girl	11 mos. later B.P. 176/114	
62	39	5	Hypertenson @ 11	164/102 @ 42	140/102	+	0	44	Spont.	6′12″ girl	4 mos. later B.P. 130/108	
63	27	14	Hypertension @ 18	140/74 @ 26	134/86	0	0	41	Med. ind.	9′ boy	?	
64	9	31	Hypertension before seen.	154/92 @ 39	132/96	+	0	40	Spont.	big boy	?	
65	35	4	Hypertension @ 15	138/106 @ 36	140/98	3+	tr.	39	Med. ind.	9′3″ girl	11 wks. later B.P. 120/90	
66	3	40	Hypertension before seen.	150/110 @ 40	152/94	+	0	43	Spont.	7′1″ boy	11 mos. later B.P. 108/80	
67	3	38	Oedema before seen.	154/96 @ 40	154/96	3+	0	41	Spont.	9′ boy	?	
68	27	12	Hypertension before seen.	140/90 @ 34	128/88	+	0	39	Spont.	8′8″ girl	?	
69	21	21	Hypertension @ 23	146/94 @ 40	148/90	4+	0	42	Spont.	6′5″ boy	7 mos. later B.P. 130/86	
70	18	22	Oedema @ 28	146/92 @ 36	138/84	2+	0	40	Spont.	6′ boy stillborn.	8 wks. later B.P. 104/80	
71	22	16	Hypertension when seen.	156/98 @ 28	140/92	0	0	38	Spont.	7′3″ boy	?	
72	16	22	Hypertension when seen.	148/106 @ 36	148/106	0	0	38	C. S.	8′9″ boy	?	
73	34	8	Oedema @ 22	144/96 @ 41	120/94	4+	+	42	Spont.	8′5″ boy	10 wks. later B.P. 112/78	
74	31	10	Oedema @ 27	112/80 @ 40	112/80	+	0	41	Spont.	6′2″ girl	?	
75	19	22	Hypertension @ 26	150/88 @ 29	128/78	0	0	41	Spont	9′2″ girl	?	
76	34	8	Hypertension @ 28	160/88 @ 18	128/88	0	0	42	Spont.	6′3″ girl	1 yr. later B.P. 130/84	
77	27	8	Hypertension before seen.	138/96 @ 35	138/96	0	0	35	Spont.	6′5″ boy		

TABLE I (*Continued*)

Case	Under My Care (in Weeks)	When First Seen (in Weeks)	First Sign of Toxaemia and When (in Weeks)	Highest Blood Pressure and When (in Weeks)	Blood Pressure at Time of Delivery	Oedema at Delivery	Albumin at Delivery	Stage at Delivery (Wks.)	Manner of Labour and Delivery	Child	Later Course	Remarks
78	25	15	Hypertension before seen.	148/96 @ 20	144/84	tr.	0	40	Spont.	8′11″ girl		
79	12	26	Oedema @ 33	130/88 @ 37	130/88	3+	0	38	Spont.	7′2″ boy		
80	36	4	Hypertension & oedema @ 32	132/100 @ 39	132/100	0	0	40	Spont.	7′6″ girl	3 yrs. later B.P. 106/66	Sterile since
81	24	14	Hypertension before seen.	190/124 @ 38	190/124	+	ft. tr.	38	Med. ind.	6′12″ girl	6 mos. later B.P. 140/90	
82	31	10	Oedema @ 16	140/86 @ 39	140/86	0	0	41	Med. ind.	9′2″ girl		
83	37	2	Hypertension @ 37	138/96 @ 38	110/68	+	0	39	Spont.	7′ girl		
84	16	25	Hypertension & oedema @27	144/94 @ 38	110/76	+	0	41	Spont.	7′8″ girl		
85	25	14	Hypertension @ 26	154/90 @ 36	142/92	0	0	39	Spont.	8′ boy		
86	20	24	Oedema @ 33	140/84 @ 42	136/76	0	0	44	Spont.	6′14″ boy		
87	30	10	Hypertension @ 14	148/94 @ 40	148/94	0	0	40	Spont.	6′14″ boy		
88	29	10	Oedema @ 19	128/94 @ 33	126/84	4+	0	39	C.S.	4′6″ girl	116/80 @ 6 wks.	Abruptio placentae. Child lived for 5 wks. then died of pneumonia.
89	30	12	Oedema @ 28	156/88 @ 39	140/90	0	0	42	Spont.	5′8″ boy	13 mos. later B.P. 120/80	
90	33	7	Hypertension @ 26	160/102 @ 40	160/102	+	0	40	Med. ind.	7′9″ boy		
91	24	8	Hypertension @ 23	158/116 @ 31	158/116	+	0	32	Med. ind.	7 mos. anencephalic with sp. bifida	136/90 @ 6 wks.	Has kraurosis vulvae at age of 33!
92	17	22	Hypertension @ 23	144/88 @ 28	144/80	0	0	39	Spont.	7′9″ girl		
93	37	3	Hypertension before seen.	136/100 @ 36	126/90	0	3+	40	Spont.	6′6″ boy	12 mos. later B.P. 116/80	Much pyelitis in pregnancy.
94	25	10	Hypertension before seen.	212/130 @ 32	190/110	3+	4+	35	bag ind.	twin girls 3′7″ 2′4″	3 mos. later B.P. 164/106	Enormous ovarian cyst removed at 2 mos. Twins lived.
95	35	3	Hypertension @ 7	144/94 @ 34	148/76	0	0	38	Spont.	7′1″ girl	4 mos. later B.P. 118/78	
96	14	26	Hypertension @ 30	150/94 @ 40	150/94	+	0	40	Med. ind.	9′6″ boy	7 wks. later B.P. 124/88	
97	16	26	Hypertension before seen.	156/100 @ 30	144/96	+	0	42	Spont.	8′1″ boy	4 mos. later B.P. 164/108	
98	35	7	Hypertension before seen.	150/98 @ 41	126/86	4+	0	42	Med. ind.	8′13″ boy		Primary inertia. Duhrssen's incisions.
99	24	6	Oedema @ 20	140/76 @ 22	120/70	0	0	30	Spont.	3′ boy—lived 4 hrs.	Still sl. pyruia 4 mos. after.	Maximal pyelitis—could not control it. Abruptio finally.
100	32	8	Oedema @ 23	136/80 @ 40	136/80	3+	0	40	Spont.	8′7″ boy		
101	26	16	Hypertension @ 32	164/104 @ 42	164/104	0	0	42	Med. ind.	mac. male	9 wks. later B.P. 156/90	
102	26	8	Oedema @ 22	140/88 @ 28	118/76	4+	0	34	Spont.	6′7″ boy		Pyelitis in this pregnancy. Severe pre-eclampsia in labour.

TABLE I (*Continued*)

Case	Under My Care (in Weeks)	When First Seen (in Weeks)	First Sign of Toxaemia and When (in Weeks)	Highest Blood Pressure and When (in Weeks)	Blood Pressure at Time of Delivery	Oedema at Delivery	Albumin at Delivery	Stage at Delivery (Wks.)	Manner of Labour and Delivery	Child	Later Course	Remarks
103	29	10	Oedema @ 26	136/86 @ 38	136/86	3+	0	39	Spont.	7'7" girl		
104	37	3	Hypertension @ 12	170/88 @ 39	170/88	0	+	40	Spont.	7'2" boy	2 mos. later B.P. 170/74	
105	23	16	Hypertension @ 18	160/100 @ 38	140/84	+	0	39	Spont.	7'7" girl		
106	36	4	Oedema @ 23	134/96 @ 37	118/86	2+	0	40	Spont.	8'2" boy		
107	4	37	Oedema before seen.	152/76 @ 40	152/76	2+	0	41	Spont.	9'3" girl		
108	20	20	Oedema @ 25	136/88 @ 37	128/76	4+	0	40	Spont.	8'9" girl		
109	31	9	Oedema @ 26	128/86 @ 37	122/76	2+	0	40	Spont.	4'10" girl		
110	28	12	Oedema @ 26	110/90 @ 38	120/80	+	0	40	Spont.	7'4" boy	.....	Pyelitis.
111	34	6	Oedema @ 21	140/86 @ 38	126/84	3+	0	40	Spont.	8'10" boy	11 wks. later B.P. 118/76	Cholecystitis after delivery.
112	35	6	Hypertension @ 12.	154/88 @ 41	128/88	+	0	41	Med. ind.	7'15" boy		
113	19	22	Hypertension @ 37.	130/92 @ 40	130/76	+	0	41	Med. ind.	8'7" boy		
114	27	12	Hypertension @ 28.	144/86 @ 30	132/74	2+	0	39	Spont.	8'8" boy		
115	11	26	Hypertension when seen.	170/90 @ 33	144/88	0	0	37	Spont.	6'7" boy		
116	32	8	Hypertension @ 20.	144/88 @ 34	150/84	0	0	40	Spont.	6'12" boy		
117	33	7	Oedema @ 32.	130/90 @ 38	130/90	+	0	40	Spont.	7'7" boy		
118	34	4	Hypertension before seen.	156/94 @ 7	130/96	+	ft. tr.	38	Spont.	7'11" boy	6 wks. later B.P. 140/82	
119	35	4	Oedema @ 28.	124/90 @ 38	124/90	3+	ft. tr.	39	Spont.	7'2" boy stillborn	5 wks. later B.P. 130/90	Prolapsed hand, version, contraction ring, craniotomy.
120	35	3	Oedema @ 24.	130/86 @ 36	130/86	2+	0	38	Spont.	8'8" boy		
121	21	18	Hypertension @ 22	140/86 @ 25	136/80	+	0	39	Spont.	6'5" girl	.....	Huge woman.
122	28	12	Hypertension @ 18	150/76 @ 34	136/86	+	0	40	Med. ind.	6'10" girl	11 wks. later B.P. 134/76	Pulmonary embolism, but survived.
123	2	36	Hypertension before seen.	136/90 @ 36	114/80	+	0	38	Spont.	child (boy) died in labour.		
124	26	14	Hypertension oedema @34	118/94 @ 37	118/94	+	0	39	Spont.	7'15" boy		
125	36	3	Hypertension @ 31	130/90 @ 33	136/84	0	0	39	Spont.	7'8" girl	.....	Pyelitis in pregnancy.
126	33	6	Hypertension @ 10	130/90 @ 34	130/84	0	0	40	Spont.	8'12" girl		
127	10	30	Hypertension @ 28	140/88 @ 40	140/88	0	0	40	Spont.	8'6" girl	6 wks. later B.P. 124/66	
128	26	12	Oedema @ 23	164/108 @ 38	164/108	+	tr.	38	Med. ind.	5'10" boy	later 6 mos. was 114/84	

Key to Abbreviations in Tables

Spont. = spontaneous delivery.
Med. ind. = castor oil, quinine and artificial rupture of membranes.
Mac. = macerated.
Wks. = weeks.
C. S. = Caesarean.
vit. = vitamin.
mos. = months.
yrs. = years.
ft. tr. = faint trace.

interest. Fully 88 per cent had no albuminuria at delivery and only 6 per cent revealed a "one plus" or more.

TABLE II

Highest B.P. attained in pregnancy	250+ / 130+	200–249 / 115–129	160–199 / 100–114	140–159 / 90–99	139 or less / 89 or less
No. of cases	1	3	24	78	22

But the most striking effect of this management is to be seen on inspecting the duration which the gestations attained and the final weight of the babies then delivered. It is only too well known how many such toxemic pregnancies, as usually managed, end in premature delivery whether artificially induced or occurring naturally, stillbirth, in abruptio placentae or in the delivery of puny "immature" infants. Such terminations are not common in this series. Excluding Cases 2, 31, 41 and 91 which ended in the induced or natural delivery of monsters, Cases 51, 52, 101 ending in miscarriages or the expulsion of macerated fetuses, and Case 94, which ended in the bag induction of twins (which survived), fully 92 per cent of these 120 pregnancies ended at term, as judged by both birth weight and known duration of gestation. Only 4 per cent of these were induced or sectioned before term. (Only one of these was sectioned—Case 88). There were only 3 per cent prematures, induced or not. There were only seven stillbirths, excluding just the gross monsters. There was only one case of classical abruptio placentae. The average birth weight, excluding the twins, miscarriages, stillbirth, and monsters, was 7 pounds 11 ounces for the boys, and 7 pounds 6 ounces for the girls.

Owing to its cost, vitamin E therapy was continued in every case only until delivery. Whether it should have been stopped then is doubtful, for some of these patients had higher or at least as high blood pressures weeks and months postpartum. This was true, too, of many who had been quite normal in the early months of their gestations, and could not have been regarded as "essential hypertensions" or "nephritic" cases.

COMMENTS

In 1937, the author[8] presented evidence to show that only about 10 per cent of the late toxemias were truly pre-eclamptic or eclamptic, having "negative" estrogen values. All the remainder could be conveniently lumped together for therapeutic purposes as belonging to the non-convulsive or high estrogen type. The toxic women presented in this series constitute the major part of all the toxic patients seen by the writer in a ten-year period. Six more patients of this type could not be included in the statistics because of scanty data. Only twenty-one women having true pre-eclampsia and three convulsive eclamptics were seen in the author's own practice (exclusive of consultations) during the same period of time. In other words, only 15 per cent of the total 158 toxemic women were really pre-eclamptic or eclamptic, which approximates our figure of eight years ago and the 9 per cent mentioned by Eastman and Whitridge.[9] Nothing in obstetrics has been either more ludicrous or more misleading than the idea that the majority of the late toxemias would progress to convulsions unless treated properly. It is not good therapy that saves 85 per cent of such women from fits, but the fact that they could not possibly have fits under ordinary circumstances. It should be noted, however, that if these women are given very large doses of vitamin E in the last day or so before labor begins when, as immediately after parturition, there appears to be a sudden, sharp fall of the blood estrogen level, convulsive eclampsia may sometimes be precipitated.[4,10] For this reason, the author now gives such women large doses of estrogens during delivery, hoping to ward off postpartum convulsions at least.

It is important to begin vitamin E therapy at least as soon as any evidences of toxemia appear. It is very much less effective if hypertension or edema becomes well established. As all of these women had blood estrogen assays when first seen, the abortion-toxemia-miscarriage taint,[11,12] was detected in nearly all, even before clinical signs developed.

It is obvious that vitamin E is not the last word in the management of these non-convulsive toxemias, but it is equally clear from this study that it should become a major item in their therapy.

If pregnancy can be regarded as a nine-months' long experiment on renal-vascular function or even on renal-vascular histopathology, the implications of this work are interesting.[1] Is there not a suggestion here of a new approach to the study of acute nephritis and nephrosis, as well as hypertension, to name the most obvious?

Finally, mention may be made of a suggestive finding in relation to the hypertensions of this non-eclamptic group of toxemic pregnancies. Several years ago the late Dr. Percy Johns, one of our pathologists, and the writer studied a series of umbilical cords obtained from fetuses aborted, miscarried, or delivered prematurely or at term. Those obtained from mothers having high blood estrogen values in pregnancy showed a notable deficiency of the elastic laminae in the blood vessel walls. These laminae were fragmented and greatly attentuated. They gave the appearance of being pulled apart, fiber from fiber. This picture often appeared early in pregnancy. It is probable that such umbilical cord vessels would convey a sluggish blood stream to the fetus unless the vis a tergo were considerably raised. May not this offer an explanation of the hypertensions of some of the group of women under consideration? Their blood pressures would need to be raised early and to considerable heights in order to nourish their fetuses properly. No form of therapy could hope to lower that blood pressure appreciably and yet preserve those fetuses. This may explain why the vitamin E used in our study has been most effective in maintaining fetal weight, and preventing maternal edema and albuminuria, and least effective in reducing maternal hypertension.

There may be some linkage between this observation on the defective structure of the umbilical vessel walls in high estrogen, low E human pregnancies, and Mason's[13] histological studies of the fetal vessels in low-E rat pregnancies. He concluded: "some still unexplained functional or structural weakness in the vascular wall, leading to dilation and stasis, is the characteristic lesion" (of E-deficiency in the fetal rat).

SUMMARY

The histories of 128 consecutive, unselected women having a late toxemia of pregnancy of the non-convulsive type are analyzed.

2. These women were usually seen for the first time in the first half of their pregnancies.

3. All but one were ambulant throughout, and engaged in their usual tasks.

4. All had "positive" blood estrogen values and were treated with vitamin E only, with or without salt restriction.

5. Seventy-nine per cent never attained a blood pressure above 160/100. At delivery 43 per cent had lower pressures than the highest attained during pregnancy.

6. Thirty-eight per cent had no edema at delivery and 30 per cent had only slight edema.

7. Eighty-eight per cent had no albuminuria at delivery and only 6 per cent had a "one plus" or more.

8. About 92 per cent of these pregnancies ended at term (this is a corrected figure). Only 4 per cent were induced or sectioned before term. There were 6 per cent stillbirths, excluding only the gross monsters.

9. The birth weight of the living boys was 7 pounds 11 ounces and of the living girls 7 pounds 4 ounces (a corrected figure).

10. This type of toxemia constitutes 85 per cent of the toxemias seen by the writer.

The writer acknowledges with gratitude a grant supplied through the courtesy of Mr. Blanc and the Hoffmann-LaRoche Company of Canada in aid of the preparation of these data.

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Eclamptic convulsions are typical in their characteristics and coma is fairly deep between the convulsive seizures. There is a general aggravation of the other symptoms of preeclampsia including increased blood pressure, edema, and renal and hepatic changes.

From "The Management of Obstetric Difficulties" by Paul Titus (C. V. Mosby Company).

PRIMARY MALIGNANT TUMORS OF THE SPLEEN

WITH SPECIAL REFERENCE TO ENDOTHELIOMAS

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PRIMARY malignant tumors of the spleen are infrequent. In 4,000 operations at the Boston City Hospital, for example, splenectomy was twice resorted to for primary malignant tumor of that organ. Theile reported four examples of angiomas of the spleen which were found in 1,900 necropsies over a period of four and a half years in the laboratory of Prof. Lubarsch. Of these only one showed evidence of malignancy.

Smith and Rusk, in 1923, summarized reports of 102 primary malignant tumors after a careful review of the literature, and added two of their own, which they designated endotheliomas.

Hausmann and Gaarde (1943) carefully studied the literature subsequent to 1923, and were able to collect forty-four additional malignant tumors of the spleen, which included nine of lymphosarcoma. Twenty additional reports were indexed from those which were not available to the authors. To this group they added nine reports collected from the records of the Mayo Clinic, thus bringing the total to 178, of which thirty-three were lymphosarcomas.

CLASSIFICATION OF SPLENIC TUMORS

In classifying splenic tumors, Smith and Rusk, as well as Hausmann and Gaarde, divided them into the following five groups:

A. *Tumors arising from splenic capsule and trabeculae:* (1) Fibroma; (2) fibrosarcoma; (3) spindle cell sarcoma. B. *Tumors arising from splenic lymphoid elements:* (1) Lymphoma; (2) lymphoblastoma: (a) lymphosarcoma, 1′ large round cell (reticular-cell variety), 2′ small round cell (lymphoblastic variety), 3′ giant lymph follicle hyperplasia; (3) Hodgkin's granuloma. C. *Tumors arising from the endothelium of splenic sinuses:* (1) Angioma; (2) endothelioma; (3) endothelial sarcoma; (4) angiosarcoma. D. *Tumors arising from cells of the reticulo-endothelial system:* (1) Gaucher's disease; (2) Niemann-Pick splenomegalia; (3) Christian syndrome; (4) reticulo-endothelioma; (5) reticulo-endothelial sarcoma. E. *Carcinoma.*

We propose, to direct attention to group C, i.e., the group of tumors which arise from the endothelial lining of the splenic sinuses, and to report another case of angiosarcoma.

ANALYSIS OF REPORTED CASES

A. *Smith and Rusk's Tumor Data.* A careful analysis of the case histories abstracted in Smith and Rusk's paper reveals that of the 104 cases, twenty-six fall into group c. Of this number, seven were classified as endotheliomas (Smith and Rusk, (two cases); Wagner; Pasinetti; Bunting; LeFort and Legueu; Temoin and Bonnel). Marshand and Aschoff, however, termed the tumor described by Wagner a large round cell sarcoma. To this group of endotheliomas, Smith and Rusk included the cases of Stengel (designated a "neoplasm of the spleen comparable to diffuse myeloma or certain infiltrating sarcomas of the liver"); Cerkasov (perithelioma); Menetrier (splenoma); Foix and Roemmele (nodular reticulosplenoma); Arnott and O'Connor (primary (?) cancer of spleen); Friedreich, Schönstädt and Gröhe (lymphosarcoma). These authors classified the tumors reported by Golovina-Ammon (angiomatous fibrosarcoma); Cesaris (fibrosplenoma); LeFort

and Legueu (endothelioma); Villard and Santy, as examples of angiomas undergoing transition to endotheliomas.

There were two tumors called endothelial sarcoma (Weichselbaum; Risel), and three angiosarcoma (Langhans; Theile; Jores).

Smith and Rusk, in their collected series of 104 primary malignant tumors of the spleen, found twenty-one examples of lymphosarcoma, most of which were considered primary in the spleen. There were also twenty-nine tumors arising from the lymphoid elements. While a large number of these were undoubtedly lymphosarcomas, either primary or secondary, those denominated small round cell sarcomas were most likely tumors of lymphoid origin, commonly called lymphomas. On the other hand, the large round cell tumors apparently arise from reticular cells and closely resemble tumors of endothelial origin often classified as splenomas. According to Smith and Rusk, eleven of the tumors included in this group were probably of endothelial origin, and were consequently included in the category of endotheliomas. There was only one example of fibrosarcoma included in this category, namely, that reported by Jepson and Albert. Risel, however, believed that this tumor arose from the sinusoidal endothelium.

In the group of miscellaneous malignant tumors of the spleen reported and culled from the literature by Smith and Rusk, were thirteen with a varied nomenclature. Several of these tumors, when studied by other investigators, were believed to be identical with neoplasms falling into other categories, such as endotheliomas, lymphosarcomas, round cell sarcomas, et cetera.

There were also nineteen tumors described as carcinoma, but Smith and Rusk were of the opinion that most of them were actually lymphosarcomas and endotheliomas. Primary carcinoma of the spleen can arise only from an embryonic inclusion of epithelial elements, and no instance of such inclusion has even been reported. It is consequently evident that the diagnosis of carcinoma must be excluded.

B. *Hausmann and Gaarde's Tumor Data* (1923–1940). This group comprised fifty-three tumors which included nine collected from the records of the Mayo Clinic.

1. Tumors arising from splenic capsule and trabeculae:
   - 9 primary sarcoma
   - 7 large round cell sarcoma
   - 1 small round cell sarcoma
   - 2 unspecified round cell sarcoma
   - 3 fibrosarcoma

2. Tumors arising from lymphoid elements of spleen:
   - 10 lymphosarcoma
   - 2 reticulum cell sarcoma
   - 4 giant lymph follicle hyperplasia

3. Tumors arising from sinusoidal endothelium:
   - 2 endotheliomas (Caldwell; Biller)
   - 1 angioblastoma (DeNavasquez)
   - 1 hemangiosarcoma (DelBuono)
   - 4 hemangio-endothelioma (Orlandi; Zeno and Cid; Jones; Paine)
   - 1 malignant hemangioma (Wright)
   - 1 sarcoma (Krumbhaar)

4. Tumors arising from reticulo-endothelial system:
   - 2 reticulo-endothelioma
   - 2 reticulo-endothelial sarcoma

5. Primary carcinoma 1 (Werwath)

Werwath's report concerned a patient who presented a nodule on the buttock. Autopsy disclosed extensive carcinoma of the spleen and liver. For the reason previously stated, the diagnosis of primary carcinoma of the spleen must here be excluded. Hausmann and Gaarde regard this as an example of metastatic carcinoma of the spleen.

FURTHER ANALYSIS OF THE GROUP OF MALIGNANT TUMORS ARISING FROM SPLENIC SINUS ENDOTHELIUM

Combining the two series of cases in the compilations of Smith and Rusk up to 1923, and that of Hausmann and Gaarde from 1923 to 1940, there are thirty-seven in all, out of a combined total of 157

primary malignant tumors of the spleen. Of these thirty-seven tumors, sixteen (43.2 per cent) were classified as endotheliomas; four (10.8 per cent) as angiomas undergoing transition to endotheliomas; four (10.8 per cent) as endothelial sarcomas; four (10.8 per cent) as angiosarcomas; six (16.2 per cent) as hemangio-endotheliomas; one (2.7 per cent) as malignant hemangioma; one (2.7 per cent) angioblastoma; and one (2.7 per cent) sarcoma. With these data as a basis, we find that at least seventeen of the thirty-seven tumors disclosed definite angiomatous features, although it appears to us that this estimate is low.

Although Ewing maintained that endothelial sarcoma constituted the most common variety of primary malignant tumor of the spleen, a review of the literature indicates that this is not the case. We are inclined to agree with Mallory's contention that most of the reported tumors fall within the category of malignant lymphoblastomas. Although some of the tumors showed definite angiomatous features, in addition to the classical sarcomatous characteristics, the greater number, approximately 54 per cent, failed to show them, but rather disclosed cellular proliferation arising directly from the endothelial cells lining the splenic sinuses. Risel, as well as Smith and Rusk, were able to demonstrate conclusively the stages of transition from the endothelial cells of the splenic sinuses to the definitive malignant cells constituting the tumor. They found that the reticular cells played no part in the formation of this group of tumors. Ribbert, however, maintained that the recognition of a tumor of endothelial origin was solely by examination during its inception, a condition which can hardly be expected to present itself. The mere presence of endothelial cells at the margin of a growth did not, according to this observer, constitute proof that the tumor arose from the splenic sinus endothelium.

Judging from the varied terminology utilized in the identification of tumors falling within this category, and from the fact that no clear cut lines of differentiation are present, except as regards the degree of vascularity, it may perhaps be expedient to place them into two groups, namely, (1) non-vascular sarcomas arising from endothelium of splenic sinuses, and (2) malignant angiomas arising from endothelium of splenic sinuses. In this manner great confusion may possibly be avoided in recording and tabulating tumors in this category.

Sex and Age Incidence. The sex was noted in 127 patients, and of these there were seventy-three men and fifty-four women. The average age was 48.5 years.

Antecedent Diseases Involving the Spleen. A positive history of malaria was given in 13 per cent of the reported cases. Syphilis was present in 4 per cent reported by Smith and Rusk, in 4.4 per cent collected by Hausmann and Gaarde, and in 33.3 per cent observed at the Mayo Clinic. There were only four patients who furnished a history of typhoid fever.

Metastases. We have attempted to analyze the subject of metastases in the group of primary malignant tumors arising from the endothelial lining of the splenic sinus based on the report of thirty-seven tumors collected by Smith and Rusk, and Hausmann and Gaarde. There were four reports in which the data were incomplete as regard the presence or absence of metastases. In sixteen reports it was definitely stated that metastases were absent. However, closer scrutiny of this group revealed that in eleven of these patients the reports were based on surgical intervention, in four on necropsies, and in one information was not given save to the effect that metastases were absent. Of the remaining seventeen patients (48.6 per cent) who came to autopsy, the liver was involved in eleven (64.1 per cent), the lung in four (23.5 per cent), lymphnodes in five (29.4 per cent), pancreas in four (23.5 per cent), and bone in four (23.5 per cent).

Even a cursory review of the survival records reveals that the general prognosis in this disease is poor, and that in many

of the patients who apparently had no metastases at the time of operation, they were undoubtedly present at the time of death. If this view is valid, it is likely that many patients had hidden metastases at the time of operation.

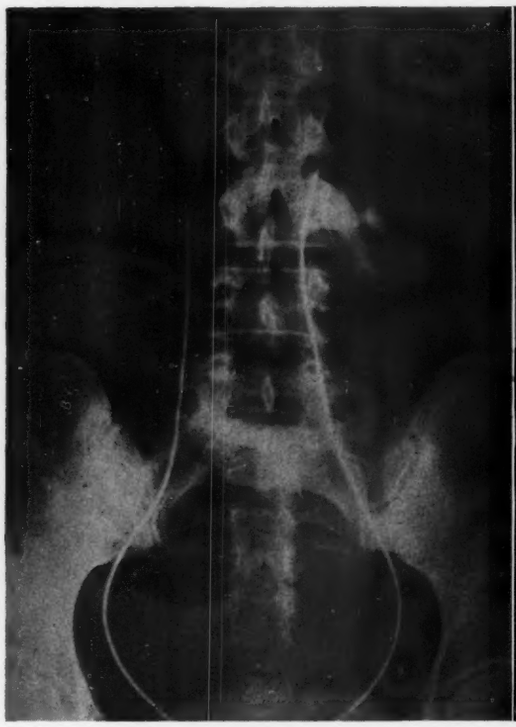

FIG. 1. Left pyelogram showing marked mesial displacement of kidney by tumor.

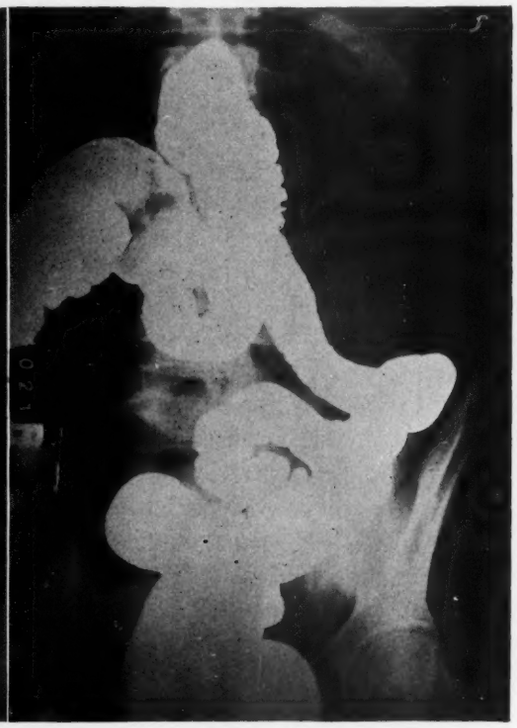

FIG. 2. Barium enema showing marked displacement of splenic flexure of colon by tumor.

Symptoms. The outstanding clinical features of this group of tumors, according to Smith and Rusk, are: (1) a rapidly growing tumor; (2) persistent or intermittent pain occasionally radiating to the abdomen or between the scapulae; (3) tenderness over the spleen; and (4) cachexia. Secondary anemia was a frequent symptom which usually occurred early in the course of the disease. Pleural effusion was present in 16 per cent and ascites in 20 per cent of the reports collected by Smith and Rusk. Spontaneous rupture of the spleen was reported in three patients (Hauptman; Bush; Kendall). Fever and leukocytosis were inconstant.

Sequelae of Splenectomy. Smith and Rusk reported that splenectomy, subtotal splenectomy or enucleation of the tumor was carried out in thirty-nine patients. Of this number, seven (17.9 per cent) died from shock, hemorrhage or other surgical complications. This high mortality can undoubtedly be attributed to faulty surgical technic in vogue in the early days of splenectomy. Thirty-two patients survived the surgical procedure, but of this number, eleven (34.3 per cent) showed the presence of metastases, local recurrence or extension on subsequent examinations, at intervals ranging from five weeks to nine years after operation. Of the patients who apparently recovered, there were twenty-one (65.7 per cent) who showed no recurrence or metastases at the time the condition was reported; but due to the fact that insufficient time had elapsed postsurgically, it is highly probable that many succumbed from extension of the tumor, recurrence or metastases.

Hausmann and Gaarde wrote that of the forty-five cases reviewed between 1923 and 1940, the spleen had been removed in eighteen. Four patients died within thirty hours after operation. Of the remaining

fourteen, two died within one year. Twelve were alive for intervals ranging between thirty days and eight years. Exploratory laparotomy showed the tumor to be inoperable in four patients. In the Mayo Clinic series of nine patients, splenectomy was carried out upon seven. One patient succumbed immediately after operation. The survival period of four others were nineteen days, three months, nineteen months, and four years, respectively. Two patients with fibrosarcoma of the spleen were found to have tumors which were inoperable.

Fig. 3. Fig. 4.

Fig. 3. Surface view of tumor.
Fig. 4. Sagittal section, showing tumor opened. Note irregular appearance of cut surface.

Histopathology. The spleen is always enlarged, often reaching enormous proportions. The tumor may be localized to a part of the splenic mass, but usually is so extensive that it completely replaces the splenic tissue. At times a thin rim of splenic pulp may be identified, but as a rule little if any normal splenic structure can be recognized when the organ is sectioned. The cut surface shows numerous whitish nodules dispersed through a reddish or bluish-red background. In the vascular tumors, ordinarily designated as angiosarcomas, many large spaces containing blood clot or soft dark-reddish, necrotic bloody material can be seen. In the solid tumors, rounded, dark-reddish, soft collapsible nodules may be noted projecting above the level of the splenic capsule.

Microscopy. Although the pulp is usually recognized in occasional small areas, most of the splenic structure is obliterated. Tumors belonging to this category, particularly angiomas, commonly show narrow channels and dilated cavities filled with blood. In some areas these vascular spaces are lined with atypical, undifferentiated cells growing rapidly and profusely. The growth of tumor cells may be so rapid and exhuberant that they are crowded together and buckle into the splenic sinuses as papillary projections. The single cell has a scanty cytoplasm, and a large vesicular nucleus which almost completely fills the cell. The chromatin, which is scanty, appears as minute granules irregularly distributed throughout the finely reticulated nucleoplasm. Nucleoli are absent as a rule, but mitotic figures are frequently found. Some parts of the tumor appear fibrous. The presence of spindle cells is not uncommon.

CASE REPORT

Mrs. C. C., aged thirty-three, married and the mother of two children, was seen December 23, 1944, because of pain in the lower abdomen and left flank, extending over a period of

several months. Two months previously she experienced a severe attack of pain in left loin accompanied by fever and weakness. Since then she had felt weak and had progressively lost weight, approximately twenty pounds. Pain in the loin never completely subsided. Although constipated all her life, this symptom increased in severity during the present illness. Anorexia was present. She noticed no blood in the stools. There were no urinary or cardiorespiratory symptoms. The past history was entirely irrelevant.

Fig. 5. Microphotograph (low power) showing remaining portion of recognizable splenic parenchyma with Malpighian corpuscles.

Fig. 6. Microphotograph (low power) showing solid syncytial tumor tissue and capillary and vascular spaces containing red blood cells (hemangio- and endotheliomatous architecture).

Physical examination showed a fairly well developed anemic woman, thirty-three years of age, who appeared chronically ill. The outstanding feature of the examination was the presence of a large tender tumor occupying the left upper abdominal quadrant. There was definite left costovertebral tenderness. The muscles of the upper abdomen were held rigidly. The liver edge was not palpable. Vaginal and rectal examinations disclosed no abnormalities. Rectal temperature was 101°F., pulse 140, blood pressure 130/80.

Cystoscopy revealed no abnormalities in the bladder, and the urinary specimens obtained from both kidneys showed no abnormal cellular elements.

Roentgenologically, a large diffuse shadow was seen filling the left hypochondrium. A left retrograde pyelogram showed the renal pelvis with a displaced upper calyx situated in close proximity to the vertebral column. The kidney proper was not enlarged, but situated directly above it there was a large diffuse shadow which appeared to be extrinsic to the kidney, but which displaced the organ downward and mesially. (Fig. 1.)

A barium enema showed narrowing of the upper part of the descending colon and broadening of the distal end of the transverse colon. The splenic flexure was displaced downward and mesially. (Fig. 2.)

Laboratory data were as follows: Urinalysis: Specific gravity 1.008; hydrogen ion concentration 5.5; albumen, faint trace; sugar, negative; microscopic 1 to 2 white blood cells per high power field. Blood Count: Hemoglobin 36 per cent; red blood cells 2,150,000; white blood cells 37,600; segmented polymorphonuclears 81 per cent, non-segmented 4 per cent, young forms 2 per cent, lymphocytes 11 per cent, myelocytes 2 per cent. The erythrocytes showed central achromia, poikilocytosis and anisocytosis. Sedimentation Rate: 34 mm. in one

hour. Tentative Diagnosis: Left infected perirenal tumor.

Operation (J.A.L.) was performed on December 27, 1944. Under gas and ether anesthesia the left loin was opened through a 12 inch Albarren incision. Extending from the under surface of the diaphragm to the pelvis, and over beyond the midline of the body, there was an enormous, soft, ovoid, lobulated tumor as large as a canteloupe, which appeared to be superimposed and partly adjacent to the left kidney. The latter organ, which appeared somewhat reduced in size, was displaced downward and mesially, but was in no way involved by the tumor mass. The splenic flexure of the colon was displaced downward. There were extensive adherent blood clots between the tumor and the attached portion of the peritoneum. The tumor, which was closely adherent to the undersurface of the diaphragm and to the lateral peritoneum, presented extensive hemorrhagic nodules directly under its capsule, giving the tumor its nodular appearance. The vascular pedicle appeared to enter the tumor on its mesial border close to the upper pole.

With considerable difficulty the tumor was delivered into the wound, and in doing so, the peritoneum was widely opened. Exploration of the abdomen failed to disclose any metastases. The pedicle of the tumor was triply ligated with chromic catgut and divided. The rent in the peritoneum was closed with a running suture of plain catgut. A rubber dam was placed beneath the diaphragm and a small tube into the renal fossa. The wound was closed in layers. One unit of plasma was administered during the surgical procedure, and 500 cc. of whole blood directly following operation. Convalescence was entirely uneventful.

The pathological report was as follows: "Macroscopic: The specimen consists of a football sized mass, measuring 25 by 15 by 11 cm. It has the shape of an enlarged spleen with a large and a small notch in one border. The surface of the mass is well encapsulated, predominantly flat, with flat undulating prominences. (Fig. 3.) There are no nodular or bosslike masses and no penetration of the surface by its contents. Parts of the surface are thin, shiny, smooth, with pinhead sized tuberclelike white thickenings. Other parts of the surface are dull, show fibrous, weblike adhesions and numerous small and larger conglomerate dense white patches. On section, splenic tissue as such is not grossly recognizable. There is, however, a thin rim of tissue in the subcapsular area which is more homogeneous and fleshy. This rim measures 1.5 cm. in its widest portion and is of a uniform blue-red hue. The remainder of the extensive cut surface consists of tissue which shows marked mottling of (a) bright and dark red, nodular, but not encapsulated, friable tissue; (b) bright red, hemorrhagic, necrotic tissue; and (c) irregular streaks and conglomerate foci of grayish white dense firm cellular and fibrous appearing tissue. These resemble anastomosing white nodules and strands upon a blue or bluish-red background, containing areas suggestive of clotted blood. (Fig. 4.)

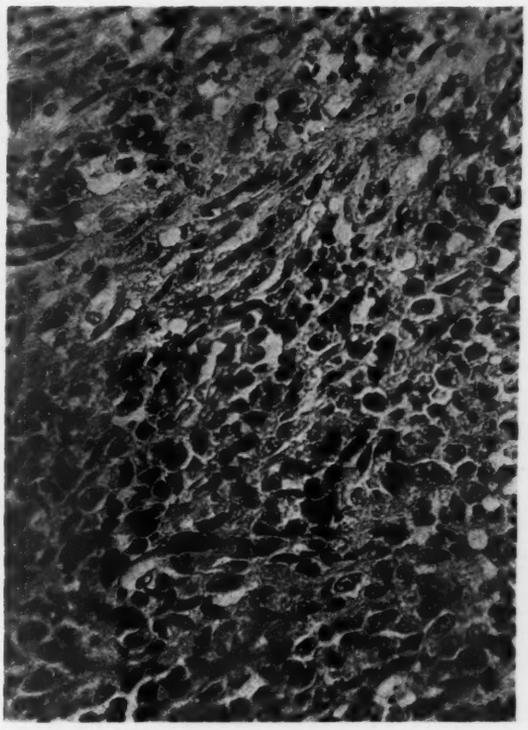

Fig. 7. Microphotograph (high power) showing solid tumor tissue with syncytial architecture, nuclear polymorphism, atypism and mitoses.

"Microscopic: Sections through the subcapsular fleshy part reveal remnants of splenic parenchyma with definitely recognizable splenic corpuscles containing a central arteriole and a mantle of dark round cells. (Fig. 5.) This mantle is of varying thickness. Elsewhere the splenic architecture is completely obliterated. This zone is replaced by abundant cellular tumor tissue and markedly hemorrhagic, cellular and fibrotic areas, and areas containing

irregular spaces filled with extravasated and clotted blood. The tumor tissue shows four types of architecture: (1) Syncitial Areas: These consist of a syncytial network of tumor cells with varying amounts of cytoplasm and nuclei varying from vesicular to dark dense and hyperchromatic. There are scattered and numerous regular and atypical mitoses. (Fig. 6.) (2) Endotheliomatous Areas: These contain lumina lined by somewhat more elongated cells, with more elongated nuclei. The lumina are small, slit-like and sieve-like. The lining cells, in places, also contain mitotic and atypical nuclei. (Fig. 7.) (3) Hemangiomatous Areas: These contain larger, irregular lumina lined by a single or multiple layer of cells varying from flat spindle-shaped and endothelial-like cells to more polygonal and syncytial cells with mitotic and atypical nuclei. The lumina are filled with blood, in places with clotted blood. (4) Fibromatous Areas: These consist of dense and loosely cellular stroma with mature spindle-shaped cells which are in continuity with the edges of the endotheliomatous and hemangiomatous areas.

"Diagnosis: Hemangio-endothelial sarcoma of spleen showing (a) extensive infiltration of splenic pulp; (b) no extracapsular infiltration."

Immediately following discharge from the hospital the patient was given a course of deep roentgen therapy. On January 8, 1945, owing to a severe secondary anemia, a transfusion of 500 cc. of citrated blood was administered. About this time she began to complain of severe pain in the region of the right knee.

Fig. 8. Femora showing metastatic tumor of right femur.

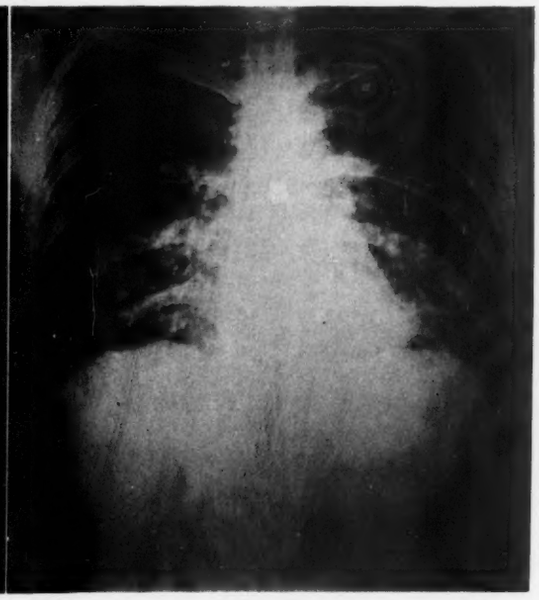

Fig. 9. Chest *x*-ray showing metastases of tha lungs.

Roentgenograms of the right femur and knee disclosed a large area of rarefaction in the medulla at the junction of the middle and upper third of the femur. (Fig. 8.) Approximately two months later she developed a persistent cough. Roentgen examination of the chest (April 13, 1945) showed massive pulmonary metastases. (Fig. 9.) At this writing the patient is confined to bed with widespread metastases.

SUMMARIZED CONCLUSIONS

Primary malignant tumors of the spleen are uncommon, but tumors taking origin from the endothelial lining of the splenic sinuses are rare (thirty-seven cases). The most frequent group of tumors of the spleen are the malignant lymphoblastomas. Although various terms have been used to identify the primary malignant tumors falling within the group arising from the endothelium of the splenic sinuses, careful examination of the morphologic features of the tumors fails to show any clear-cut differentiation between them to warrant the varied terminology. It seems to us that a great deal of confusion may perhaps be

Author	Sex	Age	Designation of Tumor	Outstanding Symptoms	Metastases	Operation	Autopsy
Smith and Rusk	M	60	Primary endothelioma	Pain left side of abdomen and back	Liver, pancreas, retroperitoneal lymph nodes, rib		Yes
Smith and Rusk	?	?	Primary endothelioma	?	?	?	?
Weichselbaum	M	21	Endothelial sarcoma	?	?	?	?
Langhans	M	30	Angiosarcoma	Pulsating abdominal tumor, pain	Liver		Yes
Wagner	F	27	Endothelioma (Marchand & Aschoff called it a large round cell sarcoma)	None	None	Splenectomy	
Pasinetti	M	47	Endothelioma	?	Liver, lungs		Yes
Albrecht	?	?	Cavernous angioma with sarcomatous changes. (Foix & Roemmele called it angiosarcoma. Smith & Rusk called it hemangio-endothelioma)	?	?	?	?
Bunting	M	40	Endothelioma	Abdominal pain, emaciation	Liver, pancreas, gastrohepatic nodes, subcutaneous tissue		Yes
LeFort and Legueu	M	56	Endothelioma. (Smith & Rusk call it angioma with transition to endothelioma)	History of malaria; large spleen, emaciation	None	Yes	
Theile	M	56	Angiosarcoma. (Risel claims this tumor similar to his own, LeFort's and Langhans')	Weakness, pain, leukocytosis, large abdominal tumor	Liver, lung stomach		Yes
Jepson and Albert	F	15	Fibrosarcoma. (Risel believes type of cell here suggests an endothelial origin)	Palpable tumor 5 months previously with dragging sensation	None	Splenectomy	
Böchelmann	?	1½	Angioma with definite sarcomatous areas	Tumor spleen	None	Yes	
Jores	F	45	Angiosarcoma	Large spleen	Liver		Yes
Risel	M	52	Endothelial sarcoma	Tumor of spleen	Liver		Yes
Temoin and Bonnel	F	27	Endothelioma	Abdominal tumor	None	Yes	
Jopson	F	33	Hemangiomatous endothelioma	Abdominal tumor and anemia	None	Yes	

Author	Sex	Age	Designation of Tumor	Outstanding Symptoms	Metastases	Operation	Autopsy
Gröhe	M	20	Lymphosarcoma (Smith & Rusk, also Bunting called it endothelioma. Risel called it lymphosarcoma)	History of previous fall followed by pain in left hypochondrium 8 months before death	Liver, mesentery, intestine, right pleura		Yes
Villard and Santy	M	22	Smith & Rusk called it angioma showing transitional changes to endothelioma	?	?	Yes	
Friedreich	M	56	Smith & Rusk, Bunting, & Weichselbaum called it endothelioma	None	Extension into diaphragm and tail of pancreas		Yes (death due to apoplexy)
Schönstädt	M	39	Mixed tumor—endothelioma and fibroma. (Smith & Rusk called it endothelioma)	Sudden onset 4 weeks before death	Right ilium dorsal vertebra, colon, stomach		Yes
Stengel	F	21	Neoplasm of spleen comparable to diffuse myeloma or certain infiltrating sarcomas of liver. (Smith & Rusk called it endothelioma)	Pain left upper abdomen tumor, loss of weight	None	Yes	
Cerkasov	M	11	Perithelioma. (Smith & Rusk called it endothelioma)	Tumor 1 year in duration	None	Yes	
Menetrier	M	47	Splenoma. (Smith & Rusk called it endothelioma)	?	Peritoneum, pancreas, suprarenal capsule, lung, lymph nodes		Yes
Golovina-Ammon	M	16	Angiomatous fibrosarcoma. (Smith & Rusk: angioma undergoing endothelial changes)	?	None		Yes (patient died of Tbc meningitis)
Foix and Roemmele	F	45	Nodular reticulo-splenoma. (Smith & Rusk: endothelioma)	Fever, anemia, cough, dyspnoea, tumor	Left suprarenal lymph nodes along splenic vein		Yes
Cesaris	F	76	Fibrosplenoma. (Smith & Rusk: angioma with slow transition to endothelioma)	Edema of legs, loss of weight	None		Yes (patient died of myocarditis)

Author	Sex	Age	Designation of Tumor	Outstanding Symptoms	Metastases	Operation	Autopsy
Arnott and O'Connor	?	?	Primary (?) cancer of spleen. (Smith & Rusk: endothelioma)	?	None	?	?
Orlandi	F	44	Primary diffuse hemangio-endothelioma	Tumor and pain in left hypochondrium	None	Yes	
Zeno and Cid	M	30	Hemangio-endothelioma	Pain in left hypochondrium and asthenia	None	Yes	
Wright	M	25	Primary malignant hemangioma	Pain in loins and back, swelling of abdomen asthenia, icterus, loss of weight	Liver	Yes	Yes
DelBuono	F	52	Hemangiosarcoma	Pain in left hypochondrium, asthenia	Cranium, spinal column	Yes	
Krumbhaar	F	75	Sarcoma of spleen	?	None		Yes (patient died of broncho-pneumonia)
Jones	F	55	Hemangio-endothelioma	Enlarged, tender spleen	None	Yes	
DeNavasquez	M	57	Angioblastoma	Pain in upper abdomen, left chest and axilla	Liver		Yes
Paine	M	64	Haemendothelioma	Abdominal pain, loss of weight, dyspnoea	Liver, bone marrow		Yes
Caldwall	M	33	Endothelioma	Pain in abdomen, nodules on trunk and neck	None		Yes
Biller	F	67	Endothelioma	Asthenia, loss of weight, anorexia, pallor	Lymph nodes		Yes

avoided if the tumors were placed in two main categories, namely, (1) non-vascular sarcomas arising from endothelium of splenic sinuses, and (2) malignant angiomas arising from the endothelium of splenic sinuses.

The pronounced symptoms are (1) a rapidly growing left upper abdominal tumor; (2) persistent or intermittent pain; (3) tenderness over spleen; (4) cachexia. These tumors are highly malignant, metastasize rapidly, and frequently involve the liver, and less frequently the lungs, regional lymph nodes and pancreas. The only treatment which offers slight hope is early splenectomy.

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SURGICAL CORRECTION OF CHIN MALFORMATIONS

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CHIN deformities are common congenital, developmental, and posttraumatic disfigurements which mar an otherwise pleasant and highly acceptable facial contour. These malformations are a detriment to the individual both socially and professionally and are often the subject for constant repeated cruel ridicule. Many unfortunates with an elongate nose and a marked underdeveloped chin have often been labeled "bird face," or "Andy Gump."

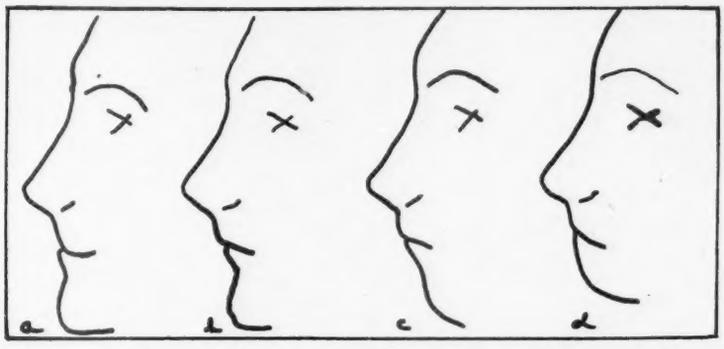

FIG. 1. Profile analysis of chin deformities: a, mild prognathism; b, normal chin; c, receded chin; d, receded and dwarfed chin.

Facial features vary considerably between and among races. There is great discrepancy among peoples as to which features are considered ideal. Individual likes and dislikes vary. Those facial characteristics which appeal to one are averse to another. When a feature is slightly over or underdeveloped, it is accepted as "going with the face." Exaggerated deformity in one or more features, however, causes an unpleasant facial appearance an embarrassment to the patient.

An accurate chart of facial alignments is impossible to establish. However, general principles have been set up for the relationship between the components which make up the face. In the white race, the most pleasant profiles are those in which the glabella and mentum are in a perpendicular line and the upper lip protrudes slightly in advance of the lower one. (Fig. 1.) Careful examination establishes the diagnosis as to what feature or features do not blend with the others. One or more may be at fault. Often, several malfeatures may be improved. In some cases, it is wiser to correct only the most marked deformity. Occasionally, when all the features are not in harmony, it is advisable not to attempt correction unless both the surgeon and patient have a good conception of the anticipated end result. This is especially important in patients with psychoneurotic backgrounds.

A chin which is hypertrophied or underdeveloped may not be a deformity in itself, but may be out of proportion in regard to the other features. (Figs. 2 and 3.) The most important features in relation to the chin are the superior maxilla and the nose. A chin which appears to be mildly underdeveloped may not be hypoplastic. The superior maxilla or upper teeth may protrude causing the jaw to appear small. Orthodentic treatment alone may be sufficient to correct the deformity. A prominent nose may cause a similar apparition. (Figs. 4 and 5.)

In planning a chin correction, it is essential to examine the patient in a completely relaxed state with the head erect, the teeth occluded, and the lips closed. The patient is carefully observed while opening and closing the mouth. When the mouth is opened, the condyle of the mandible passes forward on to the articular tuberosity of the temporal bone while the mental protuberance describes an arc downward and backward. If proper nasal ventilation is obstructed either from a nasal occlusion or enlarged nasopharyngeal vegetations, the lower jaw is displaced posteriorly, and a microgenic or "mouth breather" appearance is assumed. In these cases, simple alleviation of the obstruction causes spontaneous improvement in the esthetic appearance. (Fig. 6.) It is essential, therefore, to determine whether the deformity is actual or apparent. Receded chins are evident soon after birth. Davis and Dunn[6] advocate an adjustable form attached to the infant's bottle which they state causes decided improvement. During the growth period, the malformed chin is an orthodontic problem. The orthodontist improves, and in many cases, cures the chin deformities by restoring dental occlusion. In cases of bimaxillary protrusion where both the upper and lower teeth are protracted, the orthodontist corrects the protrusion by extracting all four first bicuspids and then retracts the protruding segments. If after restoring faulty occlusion the esthetic result is not satisfactory, as in the case of absent or hypoplastic mental tubercles or an underdeveloped mental protuberance, plastic procedures must be resorted to. The plastic surgeon's chief concern is the cosmetic end result and calls upon the more experienced orthodontist and oral surgeon for functional improvement.

A B

FIG. 2. A, preoperative photograph: humped nose and a moderately receded chin; B, postoperative photo after rhinoplasty and preserved cartilage chin implant.

Fig. 3. A and B, preoperative photographs of nasal and chin deformity; orthodontia had been performed. C and D, postoperative photographs after rhinoplasty and preserved cartilage chin implant.

A B

FIG. 4. A, preoperative photograph: prominent nose, short and receded chin; B, postoperative photograph after rhinoplasty and preserved cartilage chin implant.

A B

FIG. 5. A, preoperative photograph: prominent nose with mild recession of chin; B, postoperative photograph after rhinoplasty and small preserved cartilage chin implant.

CLASSIFICATION OF THE UNDERDEVELOPED CHIN

Hypoplastic chins may be classified as of five types: (1) Underdeveloped in a superior to inferior direction due to decreased height of the body of the mandible or absence or recession of the lower teeth; (2) underdeveloped in a lateral to lateral direction due to decreased width of the body of the mandible; (3) underdeveloped in an anterior to posterior direction due to dwarfing of the mental protuberance, absence of the mental tubercles, shortening of the horizontal rami, or a posterior dislocation of the temporomandibular joints; (4) underdeveloped unilaterally due to a unilateral first branchial arch and cleft malformation or a unilateral dislocation of the temporomandibular joint, and (5) underdeveloped in a combination of two or more of the above.

HISTORICAL DEVELOPMENT

Blair,[4] in 1909, after advancing a mandible by a bilateral section of the ascending rami for a marked retruded lower jaw, found he was unable to obtain sufficient improvement by this technic. He supplemented his operation by implanting a piece of costal cartilage together with the perichondrium under the periosteum of the chin and wired it in place. "A respectable chin resulted." Bilateral osteotomies through the ascending rami with advancement or recession of the mandible are successful in most cases. However, the potential danger of facial nerve injury causes one to substitute a simpler and as satisfactory method wherever possible. It is ideal to obtain proper occlusion of the upper and lower jaws, but a mild malocclusion is preferable to a possible facial paralysis. If a severe third degree retrusion or protrusion is present, bilateral section and advancement or recession of the mandible through the ascending ramus is justified. Thoma[42] believes that sections through the horizontal ramus are superior to those through the ascending ramus. (Fig. 7.) In mild or moderate recessions, orthodontia supplemented by a chin implant offers the simplest and safest procedure. In addition, the discomfort of having the jaws wired together for several weeks is eliminated. The literature is voluminous on reported cases of mandibular ascending rami osteotomy and ostectomy, but only a few surgeons have had extensive experience in this type of sectioning. In their hands, the operative results are good. The average surgeon, however, resorts to the simpler chin implant method.

A review of the literature reveals that successful end results have been achieved by employing various substances as chin implants to build out an underdeveloped jaw. Paraffin, celluloid, and ivory, have been used with satisfactory results but have generally been discarded. The dermis, derma-fat, derma-fat-fascia, and the crest of the illeum, are the more recent substances preferred by some surgeons. Until the advocation of preserved cartilage by O'Connor and Pierce,[31] autogenous rib cartilage and the osteocartilagenous nasal hump, septal cartilage and bone were, and still are preferred by many. They stated that, "After a five year waiting period we find that all of the refrigerated cartilage isografts that have been transplanted have retained their original size and identity." Peer[32,33] has removed preserved rib cartilage at various intervals up to two years and has proven histologically that the "dead cartilage grafts showed progressive invasion by fibrous tissue and partial absorption." Dupertuis'[7] rabbit experiments showed that, "whereas the preserved ear cartilage was actively invaded and absorbed, the costal cartilage although dead, resisted fragmentation and degeneration rather well during a period of two hundred and forty three days." Lamont[22] found little difference histologically and clinically in the structural changes between autogenous and preserved rib cartilage.

Clinical observation of preserved septal cartilage implanted under the mucoperi-

A B

Fig. 6. A, preoperative photograph: humped nose, marked deflection of nasal septum occluding both nasal passages; typical mouth breather. B, postoperative photograph after rhinoplasty and septum correction without chin implant.

A B

Fig. 7. A, preoperative photograph showing third degree chin protrusion. B, postoperative photograph eight weeks after performing a bilateral partial horizontal rami ostectomy with the coöperation of Dr. Benjamin Jacobs (oral surgeon) and Dr. Benjamin Weiss (orthodontist).

chondrium of the septum into the dorsum of the nose for saddle defects, into the scalp, eyelids and face, have given the impression that this cartilage is partially absorbed and more so under the septal mucosa.[9] Preserved rib cartilage in good size pieces, however, seemed to resist absorption and the contours remained unaltered.

A

B

FIG. 8. A, preserved rib cartilage; B, preserved septal cartilage.

The cartilage was stored in 70 per cent alcohol after removing the perichondrium. (Fig. 8.) One is advised to make repeated cultures for sterility. Repeated examinations of both the cartilage and the solution has never shown the growth of an organism. Refrigeration of the preserved cartilage has proved unnecessary. Solutions kept at room temperature for as long as three years showed no organism. Preserved rib cartilage was transplanted into the chin in sixteen cases without complication.

TECHNIC OF TRANSPLANTING PRESERVED RIB CARTILAGE INTO THE CHIN

A preoperative cast of the face for study purpose is helpful in estimating the size and shape of the proposed implant. Aufricht, in his teachings, emphasizes the value of guide lines. These are drawn by antiseptic colored dyes before the tissues are distorted by an infiltration anesthetic. Various configurations are sketched according to the deformity. For the routine symmetrical dwarfed chin the following lines may be of value: A center line is drawn vertically down the chin and submental region. (Fig. 9.) A transverse line is made in the existing sublabial sulcus or at the site of the proposed one if not present. Two vertical lines, parallel with the center line, are made marking out the proposed length of the transplant. A transverse line is made parallel to the sublabial line deliniating the width of the transplant. The thickness of the transplant is judged by placing it on the outside of the skin until the profile line is established. The last line is a small half-inch horizontal one drawn under the chin in a natural fold if

present, or on a level with the posterior border of the symphysis menti.

The cartilage is washed in fresh alcohol and saline and modeled to correct the deformity. (Fig. 10.) All sharp corners are carefully rounded. The anterior surface of the implant may be flat and not necessarily rounded as the mentum pad in most people is sufficiently thick to maintain the graceful fullness of the chin.

After local infiltration of a 2 per cent solution of procaine hydrochloride, a small half-inch submental incision is made down to the bone. A subcutaneous pocket in a plane just above the periosteum is dissected. The pocket is slightly larger than

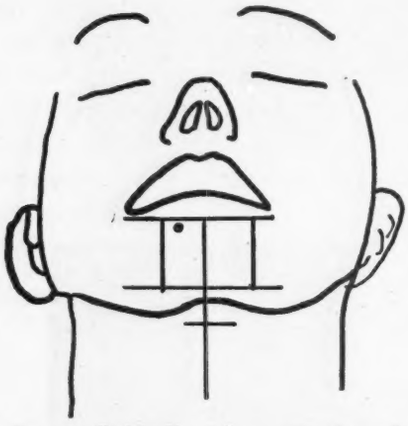

Fig. 9. Guide lines for correction of the symmetrical dwarfed chin.

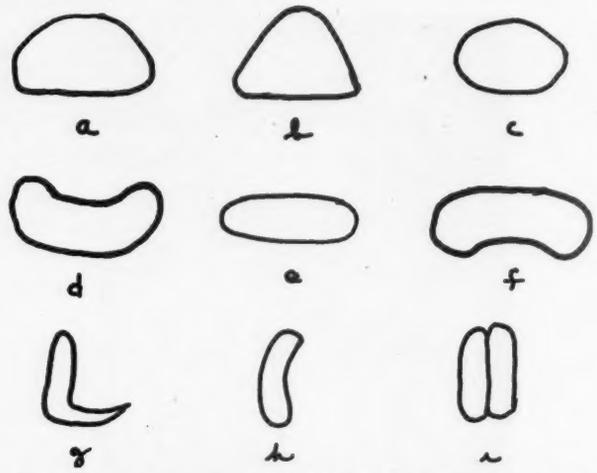

Fig. 10. a, b, c, d, e and f, full view models of various shaped implants used by the author; g, h and i, profile views.

A B

Fig. 11. A, preoperative photograph: prominent long nose associated with a mild prognathism; B, postoperative photograph after rhinoplasty alone.

Fig. 12. A and B, preoperative photograph: double chin deformity due to a submental fibrolipomatous mass; C and D, postoperative photographs following surgical remodelling.

the implant so that it may be manipulated into position. The undermining should not be superficial as the margins of the transplant may be apparent and bulge; nor should it be so extensive as to allow room for displacement of the graft. The cartilage is inserted and the incision closed by a few fine sutures. No internal fixation sutures are necessary as the cartilage becomes fixed by the surrounding tissues. A firm compression dressing is applied to maintain the cartilage in place and prevent hematomas. No drainage is permitted. The dressing and sutures are removed after the fourth day. A mild ecchymosis and a moderate edema are usually present when the bandages are removed. These disappear in a few days.

If a nasal deformity is associated with a chin deformity, correction of both results in pronounced improvement. Most patients with underdeveloped chins consult the plastic surgeon for a nasal correction when the major deformity is usually the chin. If a mild prognathism is associated with a long nose, a simple rhinoplasty without chin correction will give a gratifying result in most cases. (Fig. 11.)

Occasionally, a mass of fibrolipomatous tissue is deposited under the chin causing a double chin deformity. (Fig. 12.) It is easily corrected by employing a submental inverted T incision with the horizontal component in a natural fold. The chin is remodeled by partial subcutaneous excision of the mass. The superfluous skin is removed and the wound closed by subcuticular and fine skin sutures. Two small drains are inserted at both angles of the T and a firm compression dressing is applied to prevent the formation of a hematomas. Although an external scar persists, it is inconspicuous and the result is satisfactory.

A B

FIG. 13. A and B, "goatee nevus," nevus pilosus and pigmentosus of chin.

CONCLUSIONS

1. The methods of treatment of chin deformities have been discussed. It has been emphasized that the chin is only one of several facial features which may distort one's facial appearance. The chin should blend harmoniously with the other facial features.

2. It has been suggested that in mildly or moderately retruded chins, orthodontia combined with a chin implant is preferable to a bilateral section of the ascending rami with advancement of the mandible wherever possible.

3. Preserved rib cartilage is suggested as a suitable foreign implant which will give gratifying esthetic results when transplanted into the chin to correct a recession.

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PRIMARY RESECTION OF MALIGNANT LESIONS OF THE LARGE BOWEL*

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DUE to the many advances in chemistry and physiology it is now possible to carry out a one-stage procedure in certain cases of malignancy of the bowel, thereby avoiding the long disability and economic loss as well as the disagreeable aspect of an artificial anus on the abdominal wall.

The early attempts at overcoming obstructive lesions of the bowel date back to about 1813, and were well established at least fifty years before the development of antiseptic surgery.[1] From that date many variations in technic were recorded,[2] and in 1884 Heineke described a multiple-stage resection of the colon which was the first time that a planned procedure was suggested for carcinoma of the colon, and this procedure was divided into three stages. In 1885, Colley was the first to suggest that in resection of a tumor of the large bowel the tumor should be left *in situ* until protective adhesions occurred. Block of Copenhagen, however, was the first one who actually carried out a three-stage operation and published his results. In 1902, Mikulicz described the details of a multiple stage aseptic method of removing a tumor of the large bowel, stating that his first patient was operated upon in 1886. As a result of all the previous work the so-called Mikulicz operation was developed, although Mikulicz did not claim priority.[3]

Further progress in surgery of the large bowel consisted in the obstructive resection or in certain cases primary anastomosis preceded by an enterostomy for decompression at some distance proximal to the obstructive lesion. For a carcinoma at the rectosigmoid junction or below, an abdominoperineal resection has usually been the procedure of choice. All of these procedures necessitate the presence of either a permanent or temporary artificial anus on the abdominal wall, with its disagreeable aspect and a prolonged period of disability.

At this time further advances in our knowledge of the physiology, chemistry and pathology involved brings up the question, should a one-stage operation with primary resection be the procedure of choice in the unobstructed case with lesions down to and including the rectosigmoid area. By an unobstructed case, I mean a patient who presents no obstructive symptoms or mild obstructive symptoms which can easily be overcome by suction and liquid diet. The amount of colon removed seems to bear no relation to the hazard of operation provided attention is given to the blood supply of that portion of the bowel which remains. The site of the lesion, however, determines the amount of the bowel and mesentary to be removed along with the lymphatics. These principles are already well established. The only portion of the large bowel in which a primary resection remains questionable is in the rectosigmoid. At the present time it is generally conceded that a malignant lesion in this region requires an abdominoperineal resection and a primary anastomosis is not feasible. The objection to a primary anastomosis in this region is based on the danger of recurrence and also the hazard of infection; i.e., peritonitis, abscess, or giving way of the suture line. In the light of our present experience only one objection can be raised and that would be the question of recurrence. No matter how objectionable

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an artificial anus might be it could not compare with the tragedy of recurrence of the cancer. There are three routes in which carcinoma cells spread. (1) via blood stream, (2) direct extension, and (3) via the lymphatics. The two former present the same risk in either type of operation, so the latter seems to be the only question to be considered.

As David and Gilchrist[4] pointed out, it is relatively infrequent that we have retrograde spread of carcinoma through the lymphatics. With a primary anastomosis of the colon in the rectosigmoid area the dissection of the lymphatics can be quite as complete as though an abdominoperineal resection was performed. Therefore, the only safety factor which the latter procedure affords is the discussion of the glands in the depth of the pelvis. Certainly it must be admitted that a sharp dissection of the glands in the depth of the pelvis is far more theoretical than real.

PREPARATION

The period of preparation in the average case must be at least a week and longer if obstruction exists. Today we have well established principles of management of intestinal obstruction as outlined by Lichtenstein[5] which includes decompression by the Levine or Miller-Abbott tubes. Among the other advantages of this decompression, it leaves a bowel wall that can be safely sutured. The maintenance of fluid balance, electrolytes and proper blood levels of protein have been stressed by Meyer and Kozoll.[6]

We also have at our disposal the sulfonamides in the form of the relatively insoluble sulfasuxadine which renders the contents of the bowel relatively innocuous if spillage occurs. In addition, proper vitamin therapy is carried out, including the maintenance of normal blood level of vitamin C. Therefore, before a candidate for primary resection of the large bowel comes to surgery, the following criteria must be established:

The obstruction has been relieved by the use of suction and a liquid diet can be tolerated. The condition of the bowel wall is compatible with safe suturing, i.e., the obstruction has been relieved at least a week. The contents of the bowel have been rendered innocuous by the proper administration of sulfasuxadine over a period of four days or longer. The bowel is properly cleansed beginning forty-eight hours before surgery by multiple enemas and castor oil. Fluid and electrolytic balance have been corrected. A normal blood picture has been obtained by means of blood transfusions and plasma, amino acids, and a high protein diet. To obtain a true picture of the blood protein level and the albumin-globulin ratio, it is necessary to check the hematocrit; failure to do so will many times give one a false impression. Vitamin C is given in large doses, either hypodermically or by mouth so that a level of 1 or above is maintained at all times.

TECHNIC

The technic of primary resection of the colon with end-to-end anastomosis involves a thorough understanding of the blood supply of the large bowel. Many surgeons are reluctant to ligate the superior hemorrhoidal artery unless an abdominoperineal resection is done. However, Dixon,[7] of the Mayo Clinic, has shown that ligation of this vessel does not compromise the viability of the lower segment of the bowel which remains. A short fat mesentery limiting the mobility of the segments to be anastomosed is a contraindication for this procedure. A thorough dissection of the lymphatics must be carried out. After wide excision of the involved segment of the bowel and its mesentery the anastomosis is accomplished with interrupted fine silk or wire in two layers, any prominent vessels in the bowel wall having been individually ligated. Care must be taken that too large bites are not included in each suture because the turning in of a large cuff of bowel will predispose to undue narrowing of the lumen at the side of the anastomosis. The hole in the mesentery

that remains is sutured with plain No. (00) interrupted catgut on its two sides. A total of 2 to 4 Gm. of crystal sulfanilamide are frosted about the operative site and in different levels of the abdominal wound. In low resections, when feasible, the peritoneum is closed above the operative site. No drainage is employed.

POSTOPERATIVE MANAGEMENT

It has been shown by some investigators that extensive adverse changes take place in the blood picture during, as well as after an operative procedure of this type. It is, therefore, imperative that whole blood, plasma or intravenous fluids be administered during and following the operation, so that normal levels of the blood compliments are maintained.[8]

Postoperatively, carbon dioxide-oxygen inhalations should be given as indicated and the patient allowed to sit up early to avoid pulmonary complications.

A rectal tube must be inserted frequently in order to prevent the development of pressure on the suture line, especially in low anastomoses. A Levine tube is also employed for decompression from above.

Beginning eight hours after surgery sulfonamides are given intravenously for a period of three to four days.

COMMENTS

With these principles in mind the author has had the opportunity of resecting twenty-seven cases of carcinoma of the large bowel in the past four years, using a one-stage procedure with end-to-end anastomosis and without a preliminary cecostomy. The locations of the carcinomas were as follows: Hepatic flexure, 4; transverse colon, 6; splenic flexure, 5; rectosigmoid junction, 7; at or just above the level of the perineal reflection, 5.

The patients average stay in the hospital was eighteen days; the longest twenty-six and the shortest fourteen days. The temperature curve varied from normal to 101.2°. There was one death due to a pulmonary complication.

CONCLUSIONS

1. The period of observation in these cases is too short to evaluate the recurrence rate.

2. The technic described is not offered as an innovation but rather to demonstrate that in the light of our present knowledge of pre- and post-operative management, a primary one-stage resection of the large bowel carries with it no undue risk.

3. A primary one-stage resection is the procedure of choice for certain lesions of the large bowel rather than other methods which necessitate a prolonged period of disability accompanied by either a temporary or at times a permanent colostomy.

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GASTRODUODENAL INVAGINATION DUE TO A SUBMUCOUS LIPOMA OF THE STOMACH†

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THE differentiation of invagination of the bowel into enteric, ileocecal, and colic forms (Nothnagel, Wilms) has been considerably expanded. A complete enumeration of the forms of intussusception would include:

1. Esophageal-gastric
2. Gastric
   a. Gastro-esophageal
   b. Gastrogastric
   c. Gastro-enteric
      (1) Gastroduodenal
      (2) Gastroduodenojejunal
      (3) Gastrojejunal*
3. Enteric
   a. Enterogastric
      (1) Duodenogastric
      (2) Jejunogastric*
   b. Duodenoduodenal
   c. Duodenojejunal
   d. Jejunojejunal
   e. Jejuno-ileal
   f. Ileo-ileal
   g. Ileocecal
   h. Ileocecocolic
4. Colic
   a. Cecocolic
   b. Colocolic

Descending esophageal-gastric invaginations have as their basis mucosal hypertrophy or various tumors as cited by Henschen.[23] Clinically they may lead to a prolapse-stenosis syndrome which presents all the signs of a cardiospasm. Enderlen's account[23c,30c,45c] and that of Liaras and Ricard[29] are the only descriptions of a gastro-esophageal invagination. Both were found while dissecting anatomy cadavers and each comprised a widely dilated, pouch-like esophagus within which lay a portion of the stomach. The gastro-gastric form has been reported as such by Baylac and Dieulafé[3] and Capua[9] but probably is more frequent as a transitional phase in gastro-enteric invaginations. Engel's instance of telescoping of the stomach so that the pylorus came to lie near the cardia was an incidental finding in a cadaver and the occurrence has been interpreted as an agonal self-invagination.[23c,30c,45c] The enterogastric intussusception is an infrequent though well known complication following gastroenterostomy. The mechanism is always retrograde. The first case was described in 1917 and by 1937 forty-three similar instances were available.[5,11] Of this type acute, chronic, intermittent, and Types I to III are now current.[44] The enteric forms are by far the most common and are adequately familiar even in lesser institutions. A description of 1,000 such intussusceptions was already given by Fitzwilliams in 1908.[19] Invaginations in the colon are not too frequent and are generally grouped with the enteric forms. The ileocecal and ileocecocolic types are fairly well represented.

It is the gastro-enteric form that interests us here now. The basis for our report was provided by a patient who sought advice in 1937 and required surgery soon thereafter. After waiting eight years it still represents the only case in thirty years of a busy surgical practice; another such case of the gastro-enteric type is hardly expected.

* Forms after gastroenterostomy, the invagination occurring through the stoma.

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Bockus'[7] encyclopedic treatise describes the entity briefly and mentions only one case, that of Wade[53] in 1917. A recent

FIG. 1. Roentgengram soon after admission showing an abrupt delineation of the contrast substance traversing the middle of the stomach. A few streaks extend longitudinally. These features constitute the "abrupt sign with spurs."

supplementary review is not available. The circumstances, recommend this survey. The occurrence of the lipoma in our case affords an opportunity to summarize this form of benign gastric tumor.

CASE REPORT

The patient was a sixty-one year old, white female. When forty-nine years of age a panhysterectomy had been done (in 1925) since which she had been troubled with stubborn constipation. In 1934, she began to lose weight, was tiring easily but knew of no specific complaint. In November, 1935, she experienced a paroxysm of pain in the right upper quadrant of the abdomen which radiated through to the back and up to the right shoulder. It was accompanied by nausea and vomiting of a dark material. She was treated medically for gallbladder disease and recovered. Thereafter, she suffered other episodes of the same nature which, however, were less intense and were not associated with vomiting.

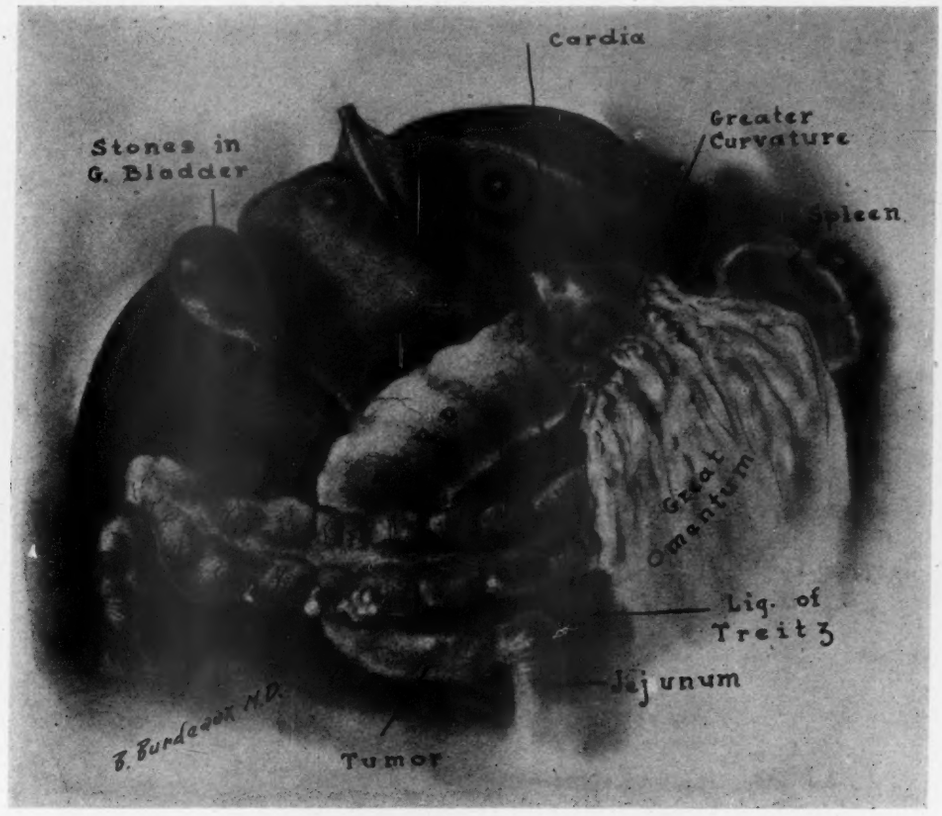
In December, 1937, the patient suddenly developed inability to retain food in her stomach. This episode had begun much like her previous gallbladder attacks. The pain was mild, but soon the vomiting became severe and prominent. It persisted for three days; whereupon, the patient was admitted to the hospital by Dr. S. M. Allerton and referred to the senior author. Her bowels had been moving; the stools were not tarry, red, nor clay colored. For about a year prior to this development she had become aware of a tumor mass just above the umbilicus. This had been growing slowly, could be moved about freely, and was not tender.

Physical examination presented a well constituted, poorly nourished woman, whose skin was dry, pale, and pasty. The abdomen was soft and not tender. A healed mid-line scar was present below the navel. A freely movable, firm mass was felt in the epigastrium and estimated to be about 3 by 5 cm. This tumor was irregular in outline and gave the impression of being located in or around the transverse colon. The remainder of the examination disclosed no other morbid conditions. The admission diagnosis was obstructive carcinoma of the transverse colon with metastatic obstruction of the pylorus.

A gastrointestinal series on the second day of hospitalization was reported as follows: The patient was too weak for a regular fluoroscopic examination, thus, only a glance was obtained. It was sufficient to disclose that the antrum of the stomach was not filling and that a retention of barium was occurring in the upper half. Chest, heart, and esophagus were normal. The regular films showed an advanced obstruction apparently involving the lower half of the stomach. (Fig. 1.) A fluid level was noted below the stomach. At six hours, the appearance was unchanged and only a very small amount of barium had left the stomach. At forty-eight hours, there was at least 50 per cent residue in the stomach and the colon was filled only in flecks. In the right flank there were eight grouped shadows each not over 1 cm. in diameter, all of which had the characteristics of opaque stones in a ptotic gallbladder. Conclusion: Obstructing lesion in the antrum of the stomach, forty-eight hour residue, obstruction of the bowel with fluid level, opaque gallbladder stones.

The laboratory data were all negative except: Hemoglobin 56 per cent, red blood cells 3,200,000; icteric index 6; urine; faint trace of albumin, few hyaline casts, much pus.

On the fifth day of hospitalization the patient developed severe, generalized pain in the abdomen accompanied by rigidity and tenderness to palpation. An enema returned dark, tarry feces. A laparotomy was performed on the ninth day.



A

FIG. 2. A, the anatomic relationships encountered during the laparotomy; B, close up of the duodenal segment.

A right paramedial incision was made extending from the costal border to the umbilicus. The right rectus muscle was reflected distally and the abdomen opened through the posterior rectus sheath and peritoneum. The pathological condition was immediately obvious: At least two-thirds of the stomach had become invaginated into the duodenum. (Fig. 2A and B.) The engaged stomach was wrinkled into broad folds but no area of puckering had developed. The uninvolved portion of the stomach was not dilated. The pylorus was evident as a thinned, pale ring of tissue and formed the proximal stoma of the intussuscipiens. The intussusceptum had pulled the greater portion of the omentum into the duodenum. The latter was dilated in its entire length and measured up to three inches in width. (Fig. 2B.) At the ligament of Treitz the distention ended abruptly. The first portion of the duodenum was elongated, under marked tension, and had rolled over the invaginated stomach. The normal horseshoe configuration was intact and the duodenum was firmly fixed; no displacement had occurred. The pancreas, bile ducts and pylorus were also in their normal topographical relationships. At the duodenojejunal junction a tumor mass could be palpated which was freely movable and was estimated to be four inches long by three inches in diameter. The gallbladder was distended and adhesions were noted between the gallbladder, duodenum and transverse mesocolon. The gallbladder contained numerous calculi.

By means of slight traction on the cardia combined with a milking manipulation retrograde toward the pylorus disinvagination could be easily effected. Considerable hyperemia and edema had developed in the duodenum, stomach, and omentum. The circulation of the disengaged bowel segments was not impaired. After total reduction of the intussusception, a large intragastric tumor could be palpated. During the manipulations the pyloric ring

rapidly regained its tonus and the duodenum contracted down to 1.5 to 2 inches. A gastrostomy was performed in the anterior wall of the stomach transversely from the lesser to the greater curvature and the contents examined. A large, pedunculated, soft tumor was disclosed attached to the posterior wall of the stomach by means of a broad, short pedicle.

Following a transient bout of atonicity of the stomach with delay in emptying as seen in a gastrointestinal series, the patient made an uneventful convalescence and was discharged from the hospital on January 15, 1938. She has been observed medically during the intervening years. There has been no symptomatology of any sort referable to the intestinal tract. In October, 1944, the patient reported for surgical check-up. Her statement was that she has not known a day of sickness since she left the hospital. She is now sixty-eight years old, has no restriction of diet and never has had to curtail any of her activities. She has gained about twenty pounds of weight which was her approximate normal weight previous to the obstructive mechanism which brought her to the doctor in 1937.

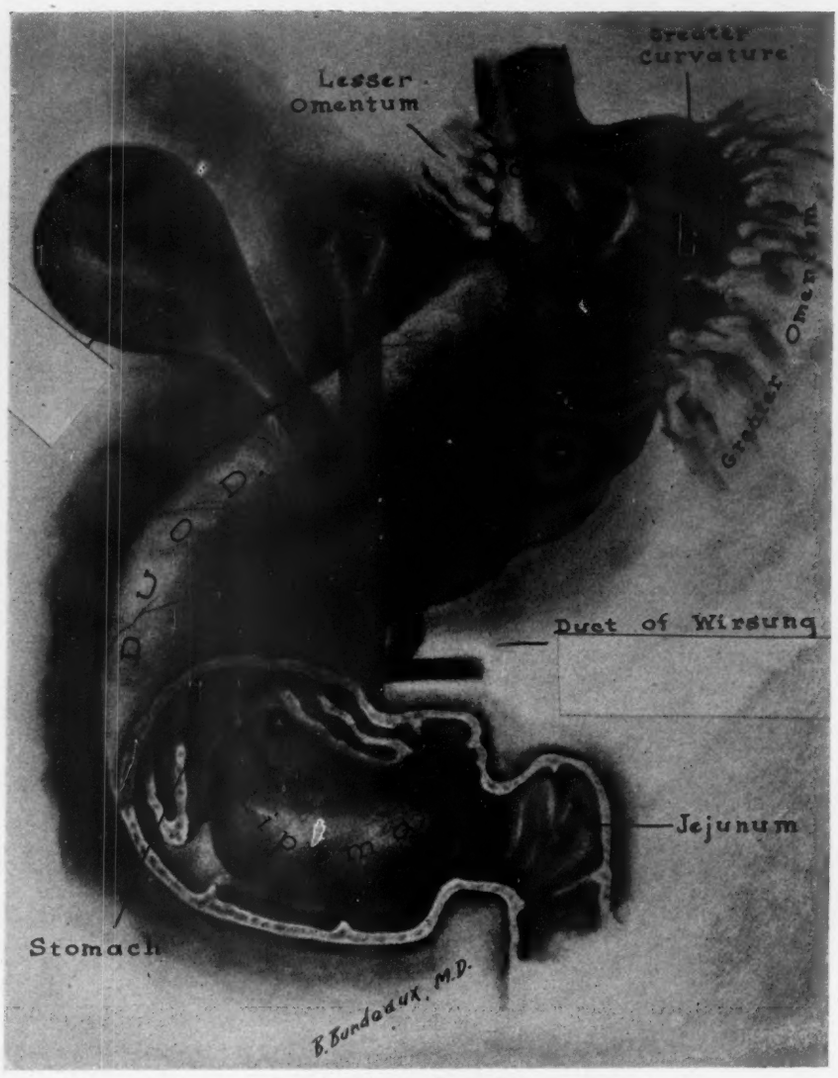
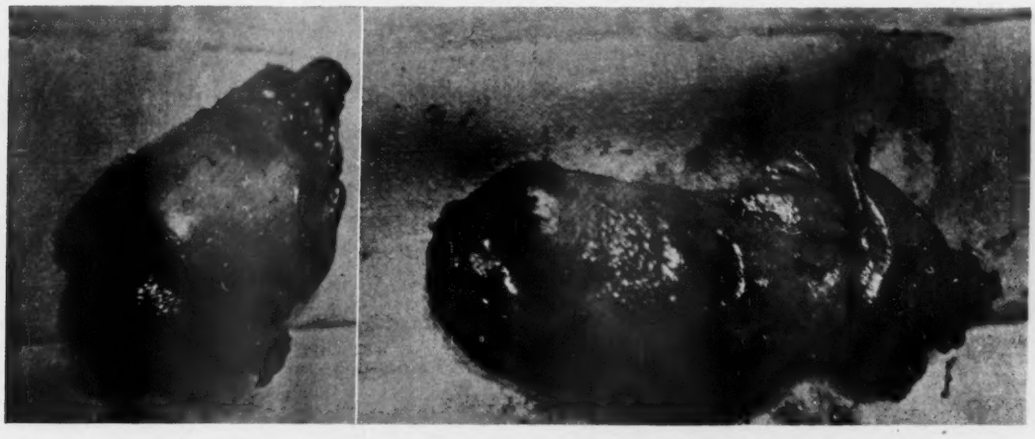


FIG. 2B. For descriptive legend see page 507.

At the time, there was uncertainty as to the nature of the tumor. In view of the broad pedicle and the absence of adhesions in the lesser omental sac which could cause interference, it was believed wiser to do a sleeve resection of the morbid portion of the stomach. The excision included wide areas of normal tissue on either side of the tumor. (Fig. 3A and B.) After the gastric anastomosis a cholecystectomy was performed in the usual manner.

Dr. V. W. Bergstrom described the specimen as follows (Figs. 3A, B and 4): "The tissue consists of a collar of stomach about 4 cm. across the lesser curvature and 8 cm. across the greater curvature. Attached to the latter and also arising from the posterior surface is a tumor 8 cm. long by 6 cm. in one diameter and 5 cm. in the other diameter. The attached end (the pedicle) is about 4 cm. in diameter, the free end has a low indentation in it. The surface of the tumor is somewhat corrugated, resembles slightly the lining of the stomach, and is markedly injected. A shallow ulcer 2.5 cm. in diameter is noted near the attachment. The tumor is very soft. On section beneath the mucosa the specimen appears to consist of a large lobule, or lobules, of pale fat.



A B

FIG. 3. Resected portion of the stomach with the tumor: A, before and B after disinvagination. A collar of stomach surrounds a soft tumor mass which is covered over by gastric mucosa. The surface is corrugated; a shallow ulcer is present near pedicle. On section the tumor is composed of lobules of fat.

"The gallbladder measures 9.2 cm. by 3.8 cm. The serosa is smooth, the wall is not thickened. On section 9 small facetted gallstones consisting almost entirely of pigment are found in the lumen. The mucosa is infiltrated with cholesterin and is rather thin.

"Microscopically, the tumor in the pyloric end of the stomach is a benign lipoma covered with gastric mucosa showing chronic inflammation and interstitial hemorrhage. Chronic cholecystitis."

EVALUATION OF THE LIPOMATOUS COMPONENT OF THE PRESENT CASE

The incidence of benign gastric tumors cannot be adequately estimated. It is supposed to be rare but Comfort[12] and Bockus[7] explain that the majority of benign tumors are too small to cause symptoms and consequently are never discovered. Even at autopsy many of the smaller intramural lesions can be overlooked and the incidence varies with the zest of the author. Root[40e] encountered seventeen benign gastric tumors in 250,000 admissions, twelve of which were proven by surgery or autopsy. Rigler and Erickson[40] in 1936 found twenty-five benign tumors of the stomach in 4,236 gastrointestinal examinations (1.6 per cent). Finesilver[21] in 1942 reported an estimated 43,200 gastrointestinal *x*-ray series studied in the period of eight years; six revealed benign tumors of the stomach. Stewart[40e,55e] surveyed 11,000 autopsies and encountered seventy-eight benign gastric tumors. Rigler and Erickson[40] reported forty-seven benign neoplasms in the stomachs from 6,242 autopsies. Rienets[40e] who was meticulous with 200 consecutive autopsies could tally thirty-two instances with one or more benign tumors of the stomach and twelve instances with cancer. Dudley, Miscall, and Morse[55] surveyed 76,077 adult surgical and medical patients admitted during three years and seven months (1935–1938). They found six benign tumors (1.3%) and 450 malignant (98.7%); 0.6% of all their admissions were treated for gastric tumors

Of 4,413 autopsies performed at Bellevue Hospital[55] during five years (1930–1934), 145 revealed tumors of the stomach (3.28%); 32 were benign, 113 were malignant.

Fig. 4. Microphotograph showing a section of the lipoma with its covering of gastric mucosa.

Some idea of the ratio of frequency between benign and malignant tumors of the stomach can be had from the following data: Eusterman and Sentry[17] reported twenty-seven benign tumors which represented 1.3 per cent of 2,168 gastric neoplasms operated at the Mayo Clinic 1907 to 1921 (e.g. 1:77). But 2,285 additional neoplasms were inoperable. This indicated that only one of 166 gastric tumors was benign. At the Lahey Clinic[27] in a period of five years the diagnosis of benign tumor was proven in six cases while the diagnosis of cancer of the stomach was made 265 times in the same period (1:44). The proportion in Rigler and Erickson's clinical material was 1:9, in their autopsy cases 1:3; Stewart's ratio computes to 1:6; Finesilver's 2:1. "The general estimate that 0.5–5% of gastric tumors are benign is too low. The figure is more probably 15–20%."[55]

Eliason and Wright,[16] Minnes and Geschickter[34] have made analyses of all tumors of the stomach as have been reported in the literature. In Table 1 these major compilations are compared. From it one can appraise the relative infrequency with which the lipoma appears as a benign gastric tumor. Records of the Mayo Clinic[12] show twenty-four submucous gastro-intestinal lipomas in a total of 3,924 necropsies, four of which were in the stomach. Eliason and Wright found one lipoma of the stomach in 8,000 necropsies. Of Stemmler's[12e] 17,000 postmortem examinations nine disclosed lipomas. Comfort[12] surveyed all reported incidences of submucous lipomas of the alimentary tract to 1931, added twenty-eight cases of his own and brought the total to 181 incidences. Of these twenty-two were in the stomach. Rumold[42] in 1941 compiled thirty-three cases of submucous lipoma of the stomach as reported in the literature during 1835 to 1940. He has sum-

Table 1
BENIGN TUMORS OF THE STOMACH
As compiled from the literature

	Eliason and Wright 1925—610 cases		Minnes and Geschickter 1936—931 cases	
	No.	Per Cent	No.	Per Cent
Myoma	325	53.2	344	36.9
Polyp	49	8.0	182	19.5
Neurofibroma	...	....	102	10.9
Papilloma	60	9.8	89	9.5
Adenoma	36	5.9	42	4.5
Fibroma	29	4.8	42	4.5
Lipoma	29	4.8	32	3.4
Polyposis	12	2.0	16	1.7
Hemangioma	10	1.6	15	1.6
Lymphadenoma	14	2.3	14	1.5
Endothelioma	...	....	12	1.2
Osteoma	1	.2	1	.1
Osteochondroma	1	.2	1	.1
Myxoma	3	.5		
Unclassified	11	1.8		
Cysts	30	4.9	38	4.1
simple	...	....	29	3.2
dermoid	...	....	5	.5
echinococcus	...	....	4	.4
	610		931	

marized each case in tabular form. Of these, seventeen were found at autopsy and sixteen had been resected at operation. The size varied from a pea to that of an orange; the sex incidence was found to be equal; the age incidence varied from 29 to 84 years. This author concluded: "Less than 1 per cent of all tumors of the stomach are benign and only about three per cent of the benign tumors are lipomas." Since Rumold's article five additional cases have appeared.[10,13,28,49,56]

In summary, the benign gastric tumor is relatively infrequent yet adequate numbers of them have been described. Of these the lipoma is the least frequent of the more important non-epithelial types. Ours is the thirty-ninth case to be reported in 120 years.

EVALUATION OF THE PRESENT INSTANCE OF GASTRODUODENAL INVAGINATION

Classification. Invaginations involving the stomach are the least frequent of the intussusceptions which are known to occur in the bowel. Henschen,[23] Vulliet,[52] and Lonnerblad[30] have in part reviewed cases reported. None is complete and enough has subsequently appeared to make a summary desirable. In Table II all available instances which have been called gastric or gastroduodenal intussusception have been compiled. They tally forty-one items. Not all, however, meet the criteria as encountered in our own case. The intussusception occurred in twenty-five women and eighteen men. The age incidence varied from twelve years to seventy-eight. In practically every instance a tumifaction was present in the stomach and could be included in the pathogenesis of the condition. These tumors comprised ten polyps, six polyposis, ten adenomas or fibroadenomas, two papillomas, six myomas or fibromyomas, one case each of sarcoma, carcinoma, hemangioma, lipoma, and neurinoma. The number of cases on the basis of mucosal hypertrophy is relatively high but could not be counted.[15,39,57,58] A case of lipoma of the stomach which herniated into the duodenum was reported by Daza and Zunega.[13] Ours is an instance of a gastric lipoma giving rise to a gastroduodenal invagination which descended to the ligament of Treitz.

All of the recorded instances represent approximately 4 per cent of the gastric tumors. Although there are still few in number, enough cases and types are available to allow for a regrouping and classification.

Firstly, an intussusception may be *complete* or *partial* depending upon whether the entire thickness of the stomach wall or only the mucosa or the tumor is involved. Schmieden[45] Pendergass,[39] Henschen,[23] Eliason and Wright[16] and more recently Eckhoff,[15] Norgore and Shuler,[57] Weinberg and Raider[58] have stressed the importance of a gastric polypositas or of a hypertrophied gastric mucosa creeping down the stomach wall to obstruct the pylorus or invaginate into the duodenum. These are *partial* forms as are also the instances of an intraluminal gastric tumor embedding itself in the pylorus or having transversed the pyloric ring eventrates into the duodenum. Fiori[20] described a polyp of the stomach that grew down into the jejunum; the gastric wall was slightly umbilicated but no invagination had occurred. In the *partial* forms only the juxtaluminal constituents enter into the condition while in the *complete* type the musculature, serosa, and the ligaments of the stomach are also involved. (Fig. 5.)

Secondly, the intussusception can be *central* or *lateral.* In the former the whole circumference of a portion of the stomach is symmetrically and uniformly pulled into a succeeding section of the stomach or into the duodenum. Therewith, the axis of the invagination coincides with or is parallel to the long axis of the stomach. In the lateral form, on the other hand, a funnel shaped puckering is present on one of the surfaces and the axis of the intussusception is at an angle to that of the stomach. (Fig. 5.)

TABLE II

Author		Reference	Sex	Age	Source of Diagnosis	Classification and Type	Tumor
Chiari	1888	23, 30, 45, 52	f	44	autopsy	lateral, complete, II	pedunculated adenomatous polyps
Collier	1896	23, 30, 52	m	21	operation & autopsy	central, complete, II	polyposis of stomach, duodenum, and jejunum
Thompson	1897	23, 52	f	75	autopsy	central, complete, II	fibroadenoma
Miodowski	1903	14, 30	....	....	.....	merely lateral indentation, no invagination	.....
Lotsch	1911	31	f	58	autopsy	central, complete, g-d-j*	myoma
Myer	1913	33	m		operation & autopsy	central, partial, Ia	polyposis
Wade	1913	53	f	48	operation	lateral, complete, g-d-j	fibromyoma
Baylac et Dieulafé	1918	3	f	43	operation	central, partial, gastro-gastric	fibromyoma
Doebler	1918	14	m	48	autopsy	central, complete, g-d-j double (see text)	myoma, mucosal polyps
Fabricus & Møller	1918	18	f	66	autopsy	lateral, complete, II	adenoma
Eusterman & Sentry	1922	17, 23, 30	f	65	operation	lateral, complete, III	fibromyoma
			f	22	operation	central, partial, Ia	hemangioma
Shuman & Cruikshank.	1923	48	f	35	autopsy	central, complete, g-d-j double (see text)	polyp, taeniae
Matas	1923	32	m	55	operation	central, partial, Ia	adenoma
Barnett	1925	2	m	54	operation	lateral, complete, g-d-j	myoma
Eliason & Wright	1925	16	m	56	operation	central, partial, Ia	mucosal prolapse
Mouat	1925	23, 30, 52	f	12	operation	central, partial, I with perforation of duodenum	adenomatous polyp
Eliason, Pendergrass & Wright, 1926; see Pendergrass, 1930.							
Vulliet	1927	52	f	51	operation	lateral, complete, g-d-j	fibroadenoma
Guleke	1927	23, 30, 52	m	32	operation	lateral, complete, Ia	adenoma
Schmieden & Westhues	1927	45	f	78	operation	central, complete, g-d-j	adenomatous polyps
Henschen	1927	23	f	51	operation	central, complete, g-d-j	mucosal papilloma
Bogoras	1930	30, 45	f	42	operation	central, complete, II	sarcoma
Pendergrass	1930	39	various		*x*-ray	herniating, prolapsing, and ball-valve types	papillomas, adenomas, fibromas, polyps; mucosal hypertrophy
Iceton, Poale, Terbutt	1931	24	f	47	autopsy	central, complete, I	neurinoma
Robson & Moynihan		24	....	....	.....	central, complete, Ia	adenoma
Saupe	1932	43	m	45	*x*-ray, operation	central, partial, Ia	polyp
			f	34	*x*-ray, operation	lateral, complete, I	papilloma
Lonnerblad	1933	30	f	45	*x*-ray, operation	central, complete, I	fibroadenoma
Pansdorf & Determan	1933	38	m	72	operation	lateral, complete, III	polyposis
Moura, de Brito, Lopes.	1934	36	m	54	operation	lateral, complete, II	fibroadenoma
			m	50	operation	lateral, incomplete, Ia	polyp
Galifi	1938	22	m	56	*x*-ray	central, partial, Ia, recedivating, transitory	no surgery nor autopsy
Fiori	1939	20	m	67	operation	lateral, partial, g-d-j	fibroadenoma
V. Meissner	1939	50	f	77	autopsy	central, complete, III	polyp
Zdansky	1939	54	f	57	*x*-ray, operation	central, complete, IV	polyp
			f	59	*x*-ray	central, complete, with external mechanism	gall stones
			m	73	*x*-ray	central, complete with external mechanism	polyp & carcinoma
Bignami	1939	6	f	69	*x*-ray	central, complete, II	no surgery nor autopsy
V. Hoyningen-Huene	1940	51	f	16	operation	lateral, complete, IV	polyp
Schmitt	1940	46	f	56	*x*-ray, operation	central, complete, I	polyp (? malig. degen.)
Rothe	1941	41	m	59	*x*-ray, operation	central, complete, I	fibroadenoma
Daza & Zuniga	1942	13	f	43	*x*-ray, gastroscopy	central, partial, Ia	lipoma
Eckhoff	1943	15	m	29	operation	lateral, partial, III	mucosal hypertrophy
Weinberg & Raider	1943	58	m	75	*x*-ray, autopsy	Central, partial, Ia	oscillating polyp
Norgore & Shuler	1945	57	f, m	61, 42	operation	central, partial, Ia	extrusion of mucosa

* g-d-j = Gastro-duoden-jejunal.

Thirdly, the severity of the invagination may be graded I to IV, depending upon the anatomic division of the duodenum reached by the intussusception. Grades I, II, III, IV successively correspond to the apex of the invaginatum having descended to the superior, descending, inferior horizontal, or ascending portion of the duodenum. Simpler instances in which the gastric tumor or the stomach mucosa (décollement) have entered the protopyloric (oral) portion of the duodenum have been called *prolapse, herniation, eventration* or *extrusion.* (Fig. 5.) Severe cases range through to the gastroduodenojejunal form.

Fourthly, Zdansky[54] Schmitt[46] amongst others, have described cases which heretofore have been called "retrograde invagination." Such a designation implies that the intussusception moved orally, e.g., opposite to the direction of peristalsis. Actually, in the recorded instances, the proximal portion of the stomach was described as descending isoperistalically and gliding over a distal segment. The ensheathing portion of the bowel in these "retrograde" cases was proximal (oral) and surrounded a distal portion of the bowel instead of being contained within the latter. It is not that an invagination occurred. The stomach had migrated forward and surrounded an adjacent section of the bowel. The direction was anterograde not retrograde. Accordingly, because the oral segment had come to lie outside of and around the aboral portion, it is suggested that this form be called *invagination with an external mechanism.* (Fig. 5.) This is in contrast to the conventional type which consists of an intussusception that arises proximal, descends,and comes to lie within an ensheathing (outer) segment of the bowel that normally is situated distally. In this more frequent form the intussusception displays an internal mechanism.

In summary, the varieties of gastroduodenal invagination can be classified as: (1) Complete or partial, (2) central or lateral, (3) invagination with an internal mechanism or invagination with an external mechanism, and (4) grade I, IA, II, III, or IV (Ia: prolapse, herniation, eventration, extrusion).

In accordance with this nomenclature our own case would be one of a complete, central invagination with an internal mechanism and Grade IV. Referring to Table II, of all the instances featured in the literature as gastroduodenal invagination, actually only two others were of this type, nine were more severe, seven were of Grade II and four were of Grade III. The central type is more frequent than the lateral, the complete more common than the partial.

Symptomatology. To date a tumor formation has always been associated with gastroduodenal invagination. As a consequence, the symptoms in the outset are those of a benign tumor of the stomach. These are numerous and variable: pain in the epigastrium or around the umbilicus, nausea, vomiting, eructations, anorexia, fatigue, diarrhea, loss of flesh, and emaciation. Hemorrhage and melena are frequent, a tumor may at times be palpated. Or the whole course of the syndrome may simulate gallbladder disease even with recurrent jaundice, the latter being caused by the tumor or the invaginatum occluding the lumen of Vater's papilla. The symptoms and the diagnosis of a lipoma of the stomach are no different from that of a benign tumor.

Gastroduodenal intussusception in its early and recidivating forms will produce many of the listed symptoms. In the late phase the clinical picture is that of an acute intestinal obstruction from which it cannot be differentiated. Intervention is an emergency and will be dictated more by the indications for operation than the diagnosis established by laparotomy.

Rather characteristic is the ball-valve syndrome which has been described with certain forms (Grades I, Ia, and II as enumerated). This syndrome is due to the sudden obstruction of the lumen of the pylorus by a prolapsed tumor, a herniation of hypertrophied gastric mucosa or

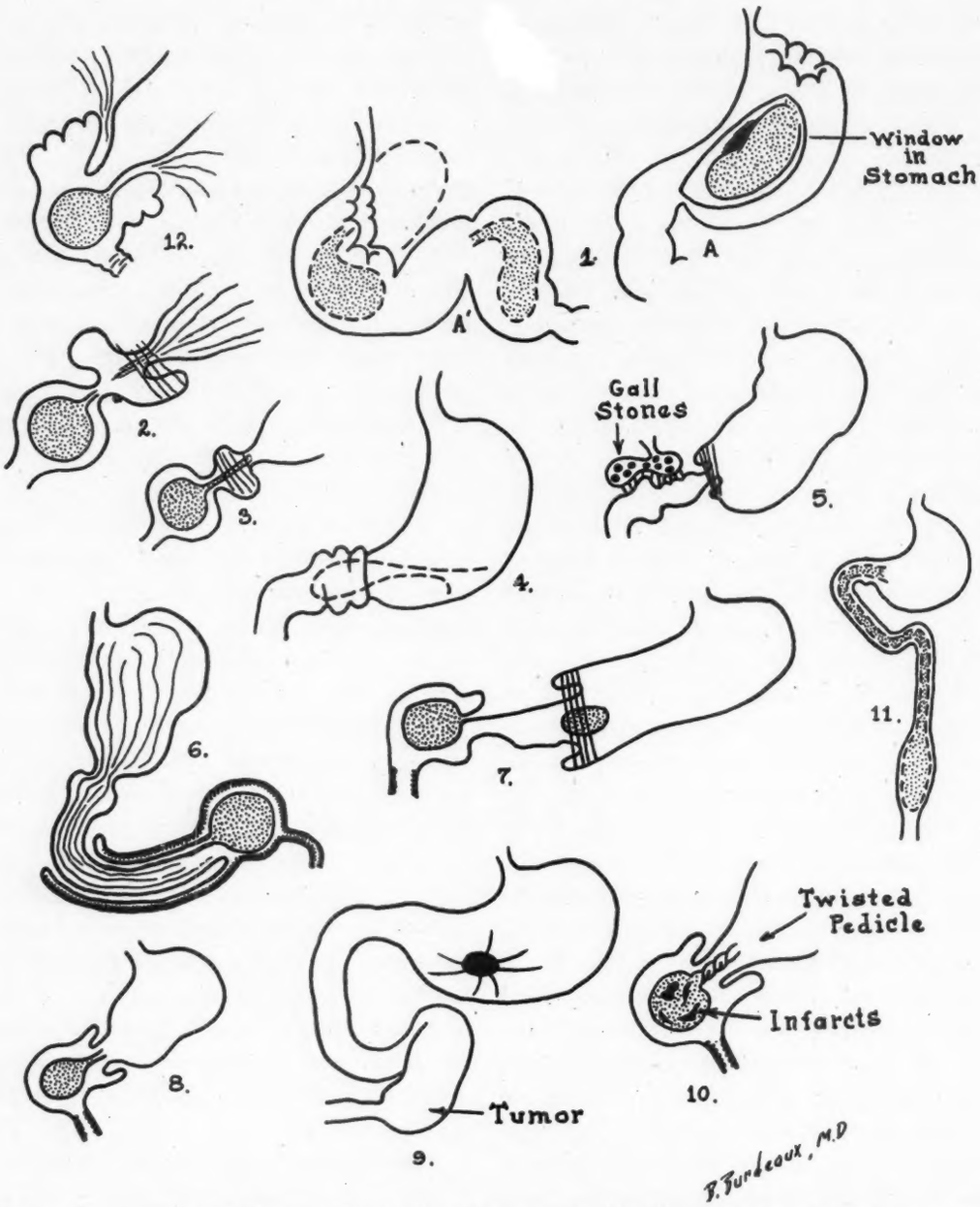


FIG. 5. A summary of features illustrating the gastroduodenal invagination: *Lateral type:* 1, showing the evolution and reaching the ligament of Treitz (Barnett); 9, with descent into the jejunum (Wade). In both, the entire thickness of the wall is involved (*complete*) and the stomach shows a funnel-shaped depression or umbilication of its surface (lateral). The axis of the intussusception is at an angle to that of the stomach. *Central type:* the whole circumference of a portion of the stomach is symmetrically pulled into a succeeding section of the bowel; the axis of the invagination is parallel to the long axis of the bowel. 4, 6, 8 and 12 represent different degrees of descent (Grades I–IV), 6 to the duodenojejunal flexure (Zdansky); *Partial Type:* only the juxta-luminal constituents of the stomach, the mucosa or the tumor, are involved. No. 11 shows a polyp that grew down into the jejunum; the outer surface of the stomach is uninvolved (Fiori). *Invagination with an external mechanism:* 5 and 7, the stomach glides isoperistaltically and ensheaths a distal segment of the duodenum instead of being contained within the latter (*internal mechanism;* 1, 4, 6). *Prolapse, herniation* of the tumor causing ball-valve syndrome is represented by 8. *Radiographic signs* (2, 3): converging axial and parallel transverse striations (Schmitt), the former due to streaks of barium within the mucosal folds in the lumen of the intussusceptum. The latter are due to the barium in the lumen of the intussuscipiens and lie external to the axial. No. 10 is the mechanism of spontaneous healing following torsion, self-amputation, and subsequent vomiting or defecation.

an early intussusception. The symptoms consist of: sudden and violent attacks of retching and vomiting accompanied by pain and flatulence. These are transitory, subside in due time but usually reappear. Between attacks the patient is comparatively well.

Radiology. Moore[35] set up criteria to facilitate the *x*-ray diagnosis of a benign tumor of the stomach. These comprise: (1) a filling defect that is circumscribed, punched-out in appearance and which is usually on the walls leaving the curvatures regular and pliant, (2) the rugae of the stomach are obliterated in the immediate area of the tumor but elsewhere they are more nearly normal in arrangement and distribution, (3) little or no disturbance in peristalsis and retention is uncommon except when the lesion is at or near the pylorus, (4) absence of a niche, neither is there any incusura or other signs of spasm, (5) close and complete approximation of the walls of the barium filled stomach. Moore pointed out that though these signs are not characteristic they are at least suggestive. Differentiation, roentgenologically, between benign tumors and other gastric lesions can seldom be absolute but in many instances the *x*-ray signs warrant an attempt at such a differentiation.

More recently the radiologists have become keen to recognize gastroduodenal invagination. Some have reported complete radiographic diagnoses before operation and Schmitt[46] has formulated features as typical for cases which are not acutely obstructed.

1. *A central area of translucency* in the bulb accompanied by transitory, moderate foreshortening of the antrum and a slight shift of the pylorus to the left at the beginning of the examination. This translucency is caused by the intussusceptum which prevents the accumulation of barium.

2. *Converging axial striations* present in the stomach and/or the duodenum. These striations (Fig. 5-(2)(6)) are longitudinal, running parallel to the long axis of the gut. They are caused by the tension created on the cardial portion of the stomach and converge towards each other as the pylorus and duodenum are reached. These axial striations represent streaks of barium within the mucosal folds in the lumen of the intussusceptum.

3. *Parallel transverse striations* running perpendicular to the axial striations but parallel to each other. (Fig. 5-(2)(3).) They are due to the barium in the lumen of the intussuscipiens and thus lie external to the converging axial striations. Both forms of striations are usually best seen in the vicinity of the pylorus. Zdansky[54] observed an instance in which the axial striations extended beyond the pylorus into the inferior horizontal portion of the duodenum and the transverse striations were in the region of the duodenojejunal flexure. (Fig. 5–(6).)

Our own case radiologically showed an abrupt delineation of the contrast medium running approximately across the middle of the stomach. (Fig. 1.) A few spurs of barium are evident extending parallel to the long axis of the stomach. These streaks, in retrospect, can now be interpreted as the equivalent of the longitudinal striations. Most of the barium was blocked sharply but enough leaked between the invagination and the stomach wall to cause the spurs. Bogara's case[30c] showed a comparable sharp delineation of the constrast medium. Saupe[43] recorded an instance that shows a linear demarcation and spurs. Bignami's case[6] at the beginning of the observation presented this "abrupt sign" but during subsequent examinations the *x*-rays showed features which though not as crass as Zdansky's are as illuminating and as typical as Schmitt's. V. Hoyningen-Huene[51] illustrated his report with a roentgengram which is almost the counterpart of our own.

In summary, radiologically, Moore's signs are in part applicable. In gastroduodenal invagination supplementary features can be encountered which consist of

converging axial and parallel transverse striations. These may be absent and in their stead the "abrupt sign" with or without spurs be available.

Therapy. A natural cure may ensue by separation of a polyp following torsion or strangulation of its pedicle. Beardsley[53c] described a case in which spontaneous detachment of a gastric polyp occurred and it was vomited by the patient. Myer[33] reported a small tumor appearing in the wash water during a gastric lavage in a case of polypositas of the stomach. Later, this patient expelled a large polyp in the feces following a hemorrhage from the bowel. Such also happened in Chosrojeff's patient.[33c] These spontaneous therapeutic successes are interesting; the rational treatment for a benign tumor of the stomach is surgery. The indications for removal, according to Kieffer,[27] become definite and absolute when obstruction, chronic blood loss or the impossibility of excluding malignancy are factors. "The weight of statistical incidence is so heavy in the direction of malignancy and the penalty for error so great that reliance upon differential findings or roentgenologic and gastroscopic examination is not feasible. The possibility of subsequent malignancy presents another indication for the surgical excision of all benign tumors."

The choice of operation varies according to different authors. Kieffer suggests a fairly wide resection of the stomach from which the tumor arises (at the cardia the difficulties would restrict the surgeon to local excision). Rumold[42] wrote: "If the presence of a benign tumor is suspected an exploratory operation and removal of the tumor with its involved wall are indicated. Removal of the stomach wall is necessary since there have been reported cases of recurrence where only the tumor was removed." Yet, Finesilver[21] recommends: "In the majority of cases of benign tumors of the stomach local excision rather than resection is indicated. The procedure most frequently used in our series was transgastric excision performed through an incision in the anterior wall of the stomach and division of the mucous membrane pedicle."

In the event of an intussusception the problem of local excision versus resection of a gastric tumor is more apt to be settled in favor of the latter. The excess amount of tissue, the severe degree of slack, the possibility of malignant degeneration, the loss of viability in the tissues surrounding the tumor would all urge resection of the involved portion of the stomach.

The rapid onset of symptoms, the picture of ileus that suddenly develops, the abrupt development of an acute condition of the abdomen all make surgery imperative in gastroduodenal invagination. The prognosis is defined by the degree of prostration, the severity of damage to the bowel, and the ability of the patient to withstand the operation. With the tissues showing no evidence of distress, disinvagination can be effected and the prolapsed side of the gastric tumor resected. Further surgery on the remainder of the bowel was not performed in any of the reported cases.

Pathogenesis. Features in the anatomy of the stomach are cited as preventatives for invagination: Its truncated configuration, fixation of the cardia and of the pylorus, fixation by the gastrohepatic and gastrocolic ligaments. The pear shape of the stomach supposedly prevents the intussusceptum being held by the intussuscipiens. Morbid features must be present for an invagination to develop and most usually it is due to a tumor of the stomach or mucosal tumifaction. These act as a foreign body within the lumen of the gut and excite the bowel to expell the tumor. Peristalsis carries it into the succeeding bowel segment and the tumor pulls the stomach after it. The tumor causes spasmodic contractions of the gut around itself but inhibition of the distal bowel segment (Wardhill[8c]). Thus the bowel is prepared to invaginate and the contracted portion with the tumor may enter the dilated segment. Chiari[45c,52c] explained intussusception on the basis of a difference in the mode of gastric contrac-

tions: Undulating in the fundus and sphincter-like in the antrum. Vulliet[52] and Thompson[52c] verified the mechanism of excitation of peristalsis associated with traction by the tumor. The tumor is always the determining factor which arouses a foreign body mechanism. Baylac and Dieulafé[3] also see the tumor as the primary factor with ptosis of the stomach, dilatation, and atonia serving as predisposing qualities.

Schmieden and Westhues[45] demonstrated on cadavers the impossibility of producing invagination when traction was applied to that segment of the stomach which is 2 to 3 cm. oral from the pylorus. In this area the traction is immediately disseminated to the lesser curvature. If, however, traction was applied 10 to 12 cm. above the pylorus the invagination could be easily induced. These authors postulated two phases in the genesis of gastric intussusception: (1) an active invagination of the stomach into the duodenum due to the traction of the tumor (or polyp); (2) a passive invagination due to a mobile duodenum gliding over the stomach after arrest of the first phase by the gastric ligaments. As prerequisite conditions for this invagination they enumerated: Frequent lesser invaginations, stretching of the tumor pedicle, gastrectasia, mobility of the duodenum, enteroptosis, and stretching of the gastrocolic ligament.

Repeated lesser invaginations frequently precede an occluding invagination. Rothe[41] followed such an oscillating instance for five years. Also others[6,22,58] speak of recidivating prolapse of the stomach or of a gastric tumor. The symptoms are transitory and vague but with engagement of the intussusception or of the tumor the aspects change rapidly. Concerning the unnatural mobility of the duodenum which facilitates the descent of the intussusception, it might be said that this feature was observed in four instances.[14,23,45,53] Generally such a finding is not recorded; neither did our case reveal defective fixation of the duodenum.

The mode of development of gastroduodenal invagination with an external mechanism is different. The stomach glides isoperistaltically and surrounds the first portion of the duodenum. Fixation of the aboral bowel segment is an absolute requirement. In one case (Fig. 5-(5)) a cholelithiasis and pericholecystitis were the cause of the fixation. In a second (Fig. 5-(7)) a prepyloric gastric tumor became impacted and anchored the bowel; the stomach propelled by a second tumor then enveloped the immobilized portion. Osedallore[37] was able to duplicate this mechanism experimentally. He infiltrated the antrum of the stomach in dogs with 0.001 per cent silver nitrate which caused a hard dense edema and elicited this form of invagination. Berman[4] has adopted this mechanism in his treatment of peptic ulcer.

Brief mention should be made of two reports of double invaginations involving the stomach. One[14] was a case of gastroduodenal invagination on the basis of a myoma and multiple polyps of the stomach. The invaginatum descended into the jejunum and at autopsy was found associated with a secondary, more distal invagination of the jejunum into itself for a distance of 8 cm. The second case[48] was an example of a gastroduodenal intussusception which in turn telescoped itself into the jejunum. The precipitating factor was a gastric polyp arising just inside the pylorus and which prolapsed into the first portion of the duodenum. This herniation acted as a plug and caused a spasm of the bowel about itself. Thereupon, the latter and the surrounding gut slipped into the jejunum. A cross section of this area disclosed jejunum, duodenum, stomach wall, and the polyp in concentric layers. At autopsy five taeniae saginata were also found to which the authors ascribed etiological significance for the origin of the second intussusception.

SUMMARY

1. A classification of invaginations as they occur in the alimentary tract is given in the introduction.

2. The history of a sixty-eight year old woman is related who experienced a gastroduodenal invagination eight years ago, the basis for which was a submucous gastric lipoma.

3. The benign tumors of the stomach are briefly reviewed (Table I) and their incidence compared with gastric cancers. Lipomas constitute about 3 to 4 per cent of the benign gastric tumors. Ours is the thirty-ninth to be reported in 120 years.

4. All instances of gastroduodenal invagination have been assembled from the literature. (Table II.) A classification of the entity is proposed. Approximately 3 per cent of the stomach tumors have invaginated.

5. Symptoms of this disease are variable and multiple. Moore's roentgenological criteria for a benign gastric tumor are mentioned and the signs of Schmitt for gastroduodenal invagination are reviewed. A supplementary "abrupt sign" is presented.

6. A tumor of the stomach or mucosal tumifaction causes a foreign body mechanism and excites the bowel to expell the tumor. The latter then pulls the stomach after itself. Predisposing factors facilitate the occurrence.

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VARICOSE VEINS

TEN YEARS' OBSERVATION ON TREATMENT

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FOR the past ten years I have been interested in the surgical treatment of varicose veins which consisted chiefly in the resection of the proximal end of the great saphenous vein, together with separate division and ligation of the uppermost tributaries of this vein, and the subsequent injection into the veins with a sclerosing solution. The technic of this operation was described by Heyerdale and myself in 1940.[1] At the same time we demonstrated the various anatomical factors that frequently brought about the recurrences of varicosities following treatment.[2] We considered that of primary importance in the recurrences was a dilatation of collateral vessels (namely, overlooked tributaries) that acted as a shunt around the site of ligation of the saphenofemoral junction. Through these shunts, hydrostatic pressure augmented by spasmodically variable intra-abdominal pressure is brought to bear on the veins distal to the site of ligation.

It is, therefore, necessary to divide and to ligate, at the proper site, not only the great saphenous vein, but also its tributaries, separately. A number of variations in the relationship of these tributaries to the parent vein were described and their importance as factors in the recurrence of the varicosities stressed. In many instances a dilated superficial femoral vein had been mistaken and ligated for the great saphenous vein. In certain cases the failure to excise a segment of the saphenous vein lead to recurrence of varicosities owing to canalization. We also stated that the injection of a sclerosing solution at the time of operation, into the portions of divided veins distal to the site of ligation, was a definite advantage. I have treated a large series (several hundred) by this method and have observed the results in many instances throughout the past ten years. All of my original observations have been repeatedly confirmed during the period, not only by myself, but by others. A few of these observations I have modified and additional observations in the treatment of varicosities have been made. It is the purpose of this paper to describe these modifications and added opinions.

USE OF THE SCLEROSING SOLUTION

Injection at Time of Operation. I have mentioned that I believed the injection of sclerosing solution at the time of the operation was exceptionally important and of definite advantage. I still believe that in the majority of cases there is an advantage in injecting a small amount of sclerosing solution at this time, the dose to be predicted on the basis of individual sensitivity test to the sclerosing agent. It is advisable to keep patients ambulatory following surgery, and in the majority of instances they are allowed to leave the hospital immediately following or shortly after surgery. Due to the number of patients who now have hospitalization insurance, it has been necessary for them to enter the hospital for economic reasons to obtain their medical insurance to which an out-patient would not be entitled. In all instances these patients are made to be ambulatory throughout their hospital stay.

There were a few cases in which the reaction to the sclerosing solution at the time of operation was much greater than anticipated, and it caused sufficient pain to necessitate bed rest. For this reason I now inject only approximately 50 per cent of my patients at the time of operation.

The 50 per cent I do not inject at this time are selected for various reasons, namely, (1) advanced varicosities associated with marked dilatation of the saphenous vein; (2) hypersensitive individuals as indicated by the response to the test dose and their reaction to the operative procedure which is performed under local anesthesia; (3) those cases in which an open ulcer or a potential ulcer exists; (4) those cases in which a previous history of deep thrombophlebitis is present, but examination revealed adequate recanalization of the deep veins; (5) pregnancy; (6) patients in whom there is already existing superficial thrombophlebitis; and (7) those cases in which segmental ligation is carried out. I believe that it is important to inject at the time of operation those patients who have only moderately advanced varicosities and have a relatively small incompetent great saphenous vein.

Postoperative Injection. I have always believed that the postoperative injection of the sclerosing solution so that complete obliteration of the varicosities occurs has been of equal importance with the operation. This has been demonstrated in a few of my cases in which it was not possible to complete treatment due to the fact that I entered the Army, or that they were already satisfied with the immediate results of the ligation and a few strategically placed injections. Although patent communicating veins had not been demonstrated in these cases prior to surgery, I am certain that in a number of instances communicating veins became incompetent and the superficial veins recanalized or persisted as varicosities. This was particularly true when the patient became pregnant and had a resulting increased intra-abdominal pressure. I believe that in these cases a back pressure occurred in the deep venous system with a resulting dilatation of the communicating veins.

COMMUNICATING VEINS

It has been my belief that in many instances too much stress has been placed on the presence or absence of communicating veins, as in most cases such situations can be handled by adequate sclerosing. This, however, is not true if a lesser saphenous incompetency exists. In some patients, particularly those who have advanced varicosities, large communicating veins can be demonstrated. These are most commonly seen in the region of the middle third of the thigh and the inner aspect of the knee. Invariably when a patient has had a deep thrombophlebitis with resulting compensatory superficial varicosities, the communicating veins will be found to have been dilated considerably. Subsequent to this compensatory dilatation, the deep venous system may recanalize and establish an adequate circulation which makes it possible to institute surgical therapy. When communicating veins of the type mentioned exist, separate ligation of the saphenous vein and the communicating veins at the demonstrated levels should be carried out. It has rarely been necessary to ligate more than one or two communicators in a single extremity, and invariably those have been in the two sites mentioned.

LESSER SAPHENOUS VEIN

Normally there is a communication between the greater and lesser saphenous veins, and in the majority of cases dilatation of a lesser saphenous vein is secondary to a valvular breakdown in the great saphenous vein. In approximately 7 per cent of my patients there has been major involvement in the lesser saphenous vein with incompetency in this system. In these instances it is advisable to ligate the lesser saphenous vein separately just as it enters into the deep venous system.

SEGMENTAL LIGATION

In individuals with advanced varicosities and markedly dilated saphenous veins, segmental division and ligation of the saphenous system is advisable regardless of the presence or absence of communicating veins. When one ligates such a

dilated saphenous vein at the saphenofemoral junction and injects a sclerosing solution into the distal portion, a hugh thrombosis with a marked phlebitis frequently occurs throughout the entire system. This causes great discomfort to the patient and may make it difficult for him to be ambulatory for a matter of a few weeks. If, however, one segmentally divides and ligates this vein and treats the individual segments in accordance with the patient's reaction, it is possible to keep the patient ambulatory and working throughout the course of treatment. In many of these cases, segments will develop localized phlebitis and become obliterated without injecting the sclerosing agent.

SUMMARY

Certain observations made in the surgical treatment of varicose veins during the past ten years have been reviewed. The injection of a sclerosing solution at the time of surgery does not seem advisable in all cases. Those cases in which this is not advisable have been mentioned. The importance of complete sclerosing obliteration of the varicosities subsequent to surgery has been stressed. When large communicating veins can be demonstrated, separate division and ligation should be done. When a hugely dilated saphenous vein is present, segmental ligation seems advisable. The importance of treatment of the lesser saphenous vein has been stressed.

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ORCHIECTOMY IN THE TREATMENT OF CARCINOMA Of THE PROSTATE*

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THE successful treatment of carcinoma of the prostate is a difficult problem. "It is progressively obstructive (50%); occurs late in life (65–80); tends toward metastasis (35%); is usually painful (75%); produces cachexia, and affects 15% of men over 50 years of age."

Early diagnosis of this disease is important for its successful treatment. The responsibility of early diagnosis falls, as a rule, upon the general practitioner, whose suspicion should become aroused when his examining finger in the rectum palpates any hard nodule or any series of irregular hard nodules in the prostate. Most patients, when first seen by the urologist, have extension of the disease beyond when the radical perineal prostatectomy of Young is indicated.

The treatment of carcinoma of the prostate by partial prostatectomy (suprapubic or perineal) or by transurethral resection has been augmented by the research work of Huggins. He reported on his treatment of prostatic carcinoma by surgical orchiectomy; Munger by irradiation of the testes. The work of both these men has revolutionized the treatment of this disease. Since Huggins' original communication there have been many reports from clinics all over the country and writers have expressed opinions varying from those of hopeful optimism to the other extreme (probably the truth lies somewhere between these extremes).

In his original reports, Huggins states that this disease is inhibited in two ways: (1) by bilateral orchiectomy which removes most of the testicular androgens; (2) by neutralizing androgenic activity. Conversely, he notes that the disease may be reactivated by injections of androgen. Inhibition of the androgens by estrogen is unsound because it is incomplete; moreover, partial inhibition is temporary and estrogen must be administered for a long time. In many species the administration of estrogen to males for a long period is in itself carcinogenic. With Huggins, we believe that bilateral orchiectomy is the method of choice as a basic treatment in advanced or metastatic prostatic carcinoma.

When metastasis begins to occur, (to the bony pelvis, lumbar vertebrae, ribs, lungs, and to the prevertebral chain of lymph nodes) there is pain due to impingement upon the nerves. There is a rise in the serum acid phosphatase (which normally is 1.5 to 3 King and Armstrong units per 100 cc. of blood) to values above 5 units when there is extension of the malignancy beyond the confines of the gland capsule. Removal of the chief source of the androgens usually produces a drop in serum acid phosphatase values, an abrupt decrease in pain, and roentgenological evidence of the disappearance of bony metastases. There is also a shrinkage of the mass of malignant tissue in the prostate, often sufficient to permit the passage of urine with more ease even when no operation has been done on the prostate.

The following is a statement from the

* From St. Vincent's Hospital, Erie, Pa.

Paul-Lewis Laboratories: "*Acid Phosphatase:* Normal values for the acid phosphatase activity of serum when determined by the method described (modified King and Armstrong technique) range around 3.25 units per 100 cc. of serum. Values falling below 5 units per 100 cc. of serum may be regarded as lying within the normal range. Values between 6 and 10 units per 100 cc. of serum are suspicious of prostatic carcinoma and should be confirmed by further determinations, over an extended period of time if necessary, and supplemented by other diagnostic procedures, cystoscopy, biopsy by transurethral or perineal routes, and roentgenologic investigation for skeletal metastases. Values over 10 units per 100 cc. of serum are diagnostic for prostatic carcinoma with metastases."

No claim is made that orchiectomy, estrogen therapy, or any combination of both of these, cures the cancer. For this reason these procedures should supplement and not supplant other indicated surgical procedures. Complete surgical removal is still the ideal treatment of prostatic carcinoma, and until more of these tumors are discovered at a stage where complete removal is possible, there can be little hope for improvement in the prognosis.

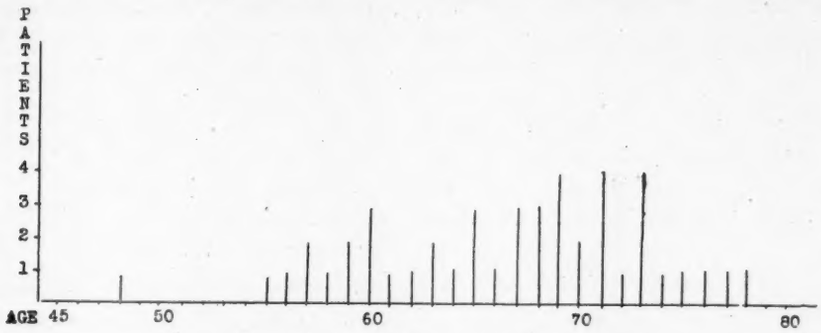

FIG. 1. Number of patients and the age groups.

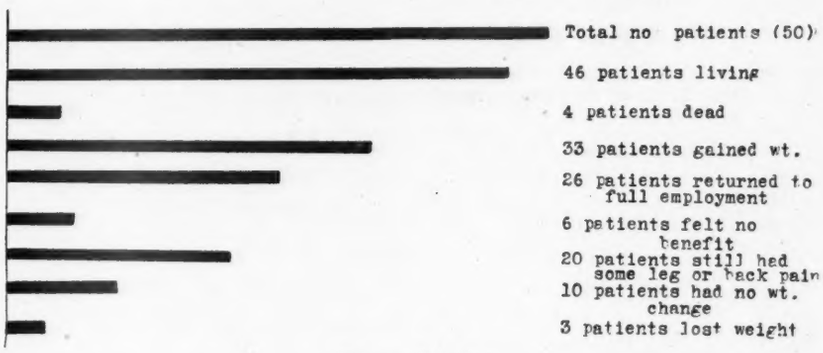
Total no patients (50)
46 patients living
4 patients dead
33 patients gained wt.
26 patients returned to full employment
6 patients felt no benefit
20 patients still had some leg or back pain
10 patients had no wt. change
3 patients lost weight

FIG. 2. Results.

We are reporting a total of fifty-four cases of carcinoma of the prostate orchiectomized at the Urological Clinic at St. Vincent's Hospital during 1941 to 1942. Forty-seven were confirmed by examination of tissue removed and only three were diagnosed by *x*-ray and serum acid phosphatase values. Eighty-eight per cent of the patients have shown definite clinical improvement of their subjective symptoms, as evidenced by their answers to the following questionnaire which was sent to the fifty patients, all of whom, fortunately, answered the query:

(Please advise yes or no and add any further details pertaining to your present complaints on the reverse side.)
Name:
Address:

1. Are you able to pass your urine normally?
2. How often do you urinate during the day_____night_____?

3. Have you had any urinary bleeding since leaving the hospital?
   When_____?
   month year
4. Is there any pain or burning on passing urine?
5. Have you gained weight? Yes_____
   No_____
6. Have you lost weight? If so, how much?
7. Has your appetite and general health improved since you were operated?
8. Do you consider yourself to be improved or unimproved?
9. Are you doing the work you did before you were operated upon?
10. Are you able to do any kind of work?
11. Have you been relieved of the symptoms of which you complained when you came to the hospital?
12. Have you had any pain involving your hips, legs or back?
13. Have you had any severe headaches before or since operation?
14. Are you undergoing any treatment for your bladder or prostate at the present time?
15. Have you had to be reoperated? If so, when and where?
16. When was your last visit to this hospital for treatment?
17. In your opinion has the operative work been a success, insofar as the relief of your symptoms and your ability to live comfortably is concerned?

This survey has shown that the average age for the occurrence of carcinoma of the prostate is 64.8 years with extremes of 48 and 78. Forty-six patients are still living (1945), four are dead of irrelevant causes. Twenty-five returned to full employment and are still working. Six patients felt no benefit. Thirty-three patients gained and have maintained their gain in weight. There was no weight change in ten patients and only three patients lost weight.

This report is being made because we believe that orchiectomy is the operation of choice in all cases of adenocarcinoma of the prostate, the exception being in those few cases that are seen early enough to do complete perineal prostatectomy (Young). We have analyzed these cases over a two-year period, giving a two- to four-year report on most of them. Study of this report shows beyond any doubt that orchiectomy with or without estrogen therapy has given us the best clinical results. We are also presenting four case reports (which are not included in this survey) because of the unusual results obtained. It is difficult for us to understand the reports of several prominent urological surgeons who condemn the procedure or at least do not think the results justify the operation. We believe with McCarthy, of New York, that if no other result but the relief of pain were obtained, the operation has merit.

The intangible causes of mental distress following orchiectomy have been almost wholly eliminated by operations devised to leave visible evidence that the contents of the scrotum still remain (Chute, Lowsley, Tolston and Hess). Orchiectomy has in some cases voided the attack upon the bladder neck obstruction. Immediate relief of obstruction, either temporary or permanent, is accomplished by resection. A certain number of cases must also receive postoperative estrogen therapy.

REPORT OF FOUR UNUSUAL RESULTS

The following four remarkable cases are reported because of the unusually splendid results which can be directly attributed to orchiectomy:

Case 1. G. W. S., age sixty-five, a white male, with the chief complaint of burning and frequency, was admitted the first time December 20, 1939. Transurethral resection of the prostate was done. Diagnosis: Adenocarcinoma, pathologically. The second admission on March 1, 1941, was made because of pain in his abdomen and difficult voiding with blood clots. We performed transurethral resection of the prostate. Diagnosis: Adenocarcinoma. *X*-ray showed no evidence of bony metastasis. The third admission on June 1, 1941, was because of urinary hemorrhage. A retention catheter was inserted and followed with bladder irrigation. On June 18, 1941, the patient was admitted again because he was unable to void. The patient had lost much weight. Suprapubic

drainage was begun; a large, soft malignant prostate and a mass in the left pelvis outside the bladder were found. The fifth admission on October 7, 1941, was made because of blood in his urine, urgency, frequency, pain over the bladder. The patient was emaciated and pale. Bilateral orchiectomy done and the suprapubic drain removed. The patient returned to the hospital to see if further surgery was necessary. The prostate is still enlarged and stony-hard, but not obstructing. No surgery was done, and there was no *x*-ray evidence of bony metastasis.

This patient is still alive (1945), has had no pain and has been able to void normally. He has had no further operative treatment, has gained weight and strength to the degree that he is able to resume his work as a chiropractor, and is able to work in his garden.

Case II. W. J. H., age seventy-one, a white male, complained of frequency, difficult and painful voiding, and the need to get up eight to ten times at night since 1939. The patient was admitted to St. Vincent's Hospital on December 11, 1942, in a somewhat psychotic state. Transurethral resection of the prostate was performed. Diagnosis: Adenocarcinoma, pathologically. There is extension of the malignancy to the bladder wall along the anterior vesical margin. Five days later bilateral orchiectomy was done, with immediate mental improvement. The patient is now employed as a machinist and has worked overtime during the past year or two. He has gained twenty or more pounds, and his nocturia is only one or two times. He has no pain.

Case III. I. W., age sixty-six, a colored male, complained of low back pain. He had been under the care of an orthopedic surgeon and in a cast for months because of a fractured vertebrae which came as the result of a fall. Due to the fact that the patient began to have voiding difficulty in the hospital, a urological consultation was had. The prostate was thought to be malignant. The patient was admitted to St. Vincent's Hospital on April 25, 1945. The serum acid phosphatase level was found to be 3 K. and A. units. *X*-ray examination showed a mottled appearance in several of the bodies of the vertebrae, with one of the vertebral bodies collapsed. A diagnosis of carcinoma of the prostate, with metastases to the spine, was made, and a bilateral orchietomy was done. There was immediate improvement in the low back pain and the ability to void normally was gradually restored. The patient is alive and well and able to work at the present time. There has been a definite gain in weight, and the patient has not had to wear a brace because of the condition of the vertebrae.

Case IV. F. L. C., age sixty-nine, a white male, complained of loss of weight and appetite. He was admitted to St. Vincent's Hospital on July 1, 1944. There had been a weight loss from 170 to 120 pounds in four months. There was no urinary complaint. Serum acid phosphatase was 24 K. and A. units on July 5th. Marked mottling of bony pelvis and lumbar spine was present roentgenographically. Bilateral orchiectomy was done followed by stilbestrol .05 mg. daily. Serum acid phosphatase was 2 K. and A. units on July 13th.

Since his operation, this patient has been relieved of his pain, has begun to gain weight, has been active again in his profession as an attorney, and is still alive (1945).

CONCLUSIONS

1. We believe, with Young, in total perineal prostatectomy for the very early case of carcinoma of the prostate. Unfortunately, we have never seen one of these cases at our clinic.

2. We believe that the proper method of treating adenocarcinoma of the prostate is to orchiectomize the patient. If there is obstruction, transurethral resection should be done. Most cases will require both procedures.

3. The report of fifty cases done in 1941 to 1942 and followed through 1945, justifies our position in the treatment of this disease.

4. Four remarkable cases are selected to illustrate the splendid improvement seen following bilateral orchiectomy and/or transurethral resection of the prostate gland.

5. We do not claim absolute cure in any patient but are more than satisfied with the relief from pain, the improvement in weight, and the ability to void in the majority of these patients.

DESICCATION OF RESIDUAL RED BLOOD CELLS FROM PLASMA PRODUCTION*

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IN a state-wide civilian blood plasma program such as that which is in operation in North Dakota,[1] large quantities of residual red blood cells are discarded as waste material. However, during the past six months these blood cells have been desiccated and distributed to physicians for use as a dressing material for various types of wound healing. The purpose of this paper is to present the technical procedure used in our laboratories for the production of dried human red cells.

Moorhead and Unger[2] presented a use for residual blood cells resulting from the mass production of plasma. They used red blood cells in the form of a gelatinous mass as a dressing material for wounds such as open ulcers, infections, etcetera.

Seldon and Young[3] used the foregoing method at the Mayo Clinic but encountered difficulty in keeping the semi-liquid material in contact with the wound. They observed that it was either absorbed by the dry gauze dressings or ran out of the wound. Because of this experience, they decided to dry the gelatinous mass and use it as a dusting powder. Dr. A. E. Osterberg, using the Harper and Osterberg[4] method for drying blood plasma, reduced the semi-liquid mass of red blood cells to a fine powder.

Seldon and Young[3] report that the powder worked satisfactorily in many instances and they cite case reports to bear this out. They recommend that the powder be dusted on the wound or applied with a sterile spatula, then covered with a dry, sterile dressing. One or two applications are used daily. The types of cases which they have treated with dried red cell powder have included infected wounds, postoperative abdominal wounds which had not healed above the fascial layer, certain proctologic cases, varicose and other ulcers of the leg, amputation stumps, open chest wounds, and so forth. They report that results have not been uniformly beneficial but were sufficiently promising to warrant more investigative work along this line.

After the report of Seldon and Young[3] was read and after several requests for red cell powder were received from local physicians, it was decided to work out a procedure for the drying of the residual blood cells for free distribution to the medical men in North Dakota. The process of desiccation is essentially the same as that used in the preparation of dried blood plasma. The method and apparatus used in the drying of the red cells is that devised by Strumia and McGraw[5] for the dehydration of plasma. This method and apparatus is being used in the preparation of blood plasma for state-wide distribution. The apparatus† does both shell freezing and drying of material, is easy to operate, and is economical to run.

LABORATORY PROCEDURE

Pooling. Blood to be used for plasma is collected in a 650 ml. rubber-stoppered bottle containing 50 cc. of saline-citrate solution. The plasma is separated from the red blood cells by centrifuging for one hour at 2,500 r.p.m. The plasma is then

† Constructed by Precision Scientific Co., of Chicago, and distributed through A. S. Aloe Company, St. Louis, Missouri.

* From the Division of Public Health Laboratories, North Dakota State Department of Health, Grand Forks, North Dakota.

aspirated from the packed cells, which remain in the lower half of the bottle.

These remaining cells are removed by aspiration into a four-liter bottle, each pool containing between 3,500 to 4,000 cc. The pooling is done in a sterility room and is handled in a manner similar to that used in pooling plasma. The cells are filtered through a gauze filter contained within the pooling bottle. The cells are thoroughly mixed by gentle agitation and are allowed to stand at room temperature for about four hours, after which cultures are made to determine sterility. Approximately 5 cc. of cells are seeded in a 30 cc. rubber-stoppered serum bottle containing 20 cc. fluid thioglycollate medium.

After the initial culturing, a 1:50,000 dilution of phenyl mercuric borate is added as a preservative. The pools are then placed in a refrigerator for approximately forty-eight hours before being dispensed. At the end of forty-eight hours the first cultures are checked and a second set of cultures is prepared at the time of dispensing.

Dispensing. The cells are dispensed by positive pressure directly from the pool into the bottle which is used for drying. The cells must be diluted before shell freezing prior to desiccation can be accomplished. Since all the plasma has been removed, the cells are a heavy thick mass; therefore, 50 cc. of cells is dispensed into a sterile rubber stoppered 400 ml. round bottle containing 200 cc. normal saline solution.

Shell Freezing. The procedure followed in shell freezing the cells is that outlined by Strumia and McGraw[5] and used in our laboratories for shell freezing of plasma. The cells are mechanically shell frozen by rotating the bottle (½ to 1 r.p.m.) with 12 mm. immersion in alcohol cooled to minus 30°C. The time required for this procedure is between forty-five minutes and one hour and with the machine we are now using twelve bottles can be frozen every hour.

When the cells are frozen, the bottles are placed in a storage cabinet at minus 20°C. until they are to be desiccated.

Drying. The standard apparatus[5] used can dry twenty-four bottles of red cells every twenty to twenty-two hours. Drying of the red cells from the frozen state is accomplished by low temperature water condensation in vacuo.

The dried plasma obtained with this apparatus is a light, porous material, with a distinct crystalline structure and may readily be reduced to a fluffy powder by shaking the bottle. On the other hand, the red cells, though they are porous and have a crystalline structure after drying, cannot be reduced to a powder through shaking. They must be removed from the bottle, placed in a sterile mortar and ground to a fine powder. This process is carried out in a sterility room with all necessary aseptic technic. As soon as the material has been reduced to a fine powder, it is dispensed into 4 ounce sterile powder jars with screw caps.

The final product is a rusty brown fine porous powder, containing not more than 1 per cent moisture, readily soluble in water. When re-dissolved in normal saline solution, smears stained with Wright's stain show no definite cellular structure, the red cells, as one would expect, are completely disintegrated during the drying process.

CLINICAL USAGE

The dried red cells, as produced with the described procedure, have been used with success. The material was sent to a few private practitioners to determine whether or not production of this product was feasible. Since then requests for these dried cells have been received from other physicians. Reports received on the usage have been very gratifying. One physician writes that he used these cells on a chronic indolent ulcer of the leg with remarkable progress in clearing up of infection and in epithelization. Another report received was on a very severely ulcerated ulcer of the leg with infection;

dried red blood cells improved the condition much and healing was rapid. Other reports have indicated the use of dried cells on indolent ulcers, slow healing processes of various types and on burns. This treatment has appreciably hastened the healing of all cases in which it has been used.

Dried red cells are now being distributed free of charge to all physicians and hospitals in North Dakota. Accumulated reports may give interesting data concerning their use and may lead to the further development of a better product.

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Hemostasis is essential to primary wound healing. The formation of a hematoma within a wound prevents coaptation of the wound margins, prolongs cicatrization, and predisposes to infection. If large clots develop in wounds, evacuation may be necessary; but opening a wound to remove blood or serum invites infections.

From "Operations of General Surgery" by Thomas G. Orr (W. B. Saunders Company).

ACROMIOCLAVICULAR JOINT INJURIES

A MODIFIED CONSERVATIVE FORM OF TREATMENT

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IN our experience, the most frequent injury to the shoulder joint, which is incurred during infantry combat training, is that to the acromioclavicular joint. This injury is usually the result of a direct downward and forward blow upon the upper portion of the scapula. A forward somersault over a small obstacle should be executed with a roll and thus avoid any direct trauma to one particular area. Occasionally the soldier slips and instead of rolling, he lands on the "top of the shoulder." The sudden shock is then absorbed in the area of the dorsal aspect of the acromion and the spine of the scapula. The force tends to push the scapula downward and forward.

The articular surface of the acromion faces upward, inward and forward against the outer aspect of the clavicle. A downward, forward and outward force on the scapula tends to shear the articular surface of the acromion away from the clavicle in its forward plane.

There are two strong ligaments which resist this shearing motion: (1) The superior and inferior acromioclavicular ligaments which surround this small joint; (2) the coracoclavicular ligament which is divisible into two parts, viz., the conoid and trapezoid ligaments.

The acromioclavicular ligament, being the weaker, gives way more readily. An injury of moderate severity results in a sprain of this ligament. A more severe injury may rupture part or all of the fibers composing the acromioclavicular ligament and result in variable degrees of subluxation. A very severe injury may also rupture the coracoclavicular ligament, either partially or completely. Hence, the extent of sprain or rupture varies directly with the severity of the trauma.

The downward pull on the acromion is exerted by the weight of the upper extremity plus the pull of the pectoralis major and the latissimus dorsi. The upward pull on the clavicle is exerted by the upper fibers of the trapezius. Both forces tend to maintain a strain on traumatized ligaments or a separation of partially ruptured ligaments.

The largest number of these injuries which we encountered were those varying from a severe sprain to a partial rupture of the acromioclavicular ligament. Most of these cases showed no evidence of subluxation. About 35 per cent showed variable degrees of mild subluxation.

It was believed that any form of fixation which would hold the clavicle down and push the acromion up would force the surfaces of the acromioclavicular joint together. This would aleviate a constant strain on the contused or partially ruptured ligaments and promote healing.

Since most of our injuries were severe sprains or those with a minimal degree of subluxation at the acromioclavicular joint, operative intervention appeared unnecessary. An attempt was made to secure some form of conservative treatment which would be practical, easy to apply and answer the purpose from a mechanical point of view.

Thorndike and Quigley,[1] Hart,[2] Shaar,[3] Benson,[4] Copher,[5] Trynin,[6] Mercer,[7] Key and Conwell[8] and many others have all described various modifications of conservative treatment for this type of injury. The author claims no originality for this method of treatment, for in reality, it is a modification of that used by many other surgeons.

A three-inch roll of adhesive, two soft felt pads measuring 3 inches by 2 inches by ½ inch, any skin glue or adherent, and one pound bag of weight is all that is necessary. The area of the back, shoulder

and forearm to be covered by the adhesive are first painted with the skin glue. This prevents slipping of the adhesive and the back, to end close to the area where the first adhesive plaster was started. A one pound bag of weight is then strapped

Fig. 1. Front view of sling applied to the patient.

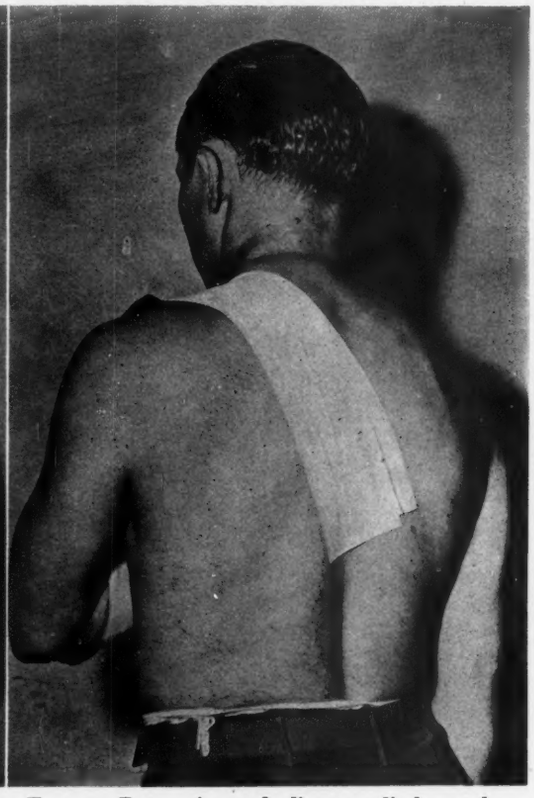

Fig. 2. Rear view of sling applied to the patient.

protects the skin. One pad is placed over the distal end of the clavicle. The other pad is placed over the ulna, just distal to an imaginary line dropped from the inner border of the biceps brachii when the elbow joint is flexed to a right angle. (Fig. 2.) It is not necessary to exert any tension on the adhesive. The first strip is carried over the upper pad, down to the flexed forearm. (Fig. 3.) The second strip of adhesive is started just distal to the upper pad, face to face with the first, to which it is adhered. The forearm is held at about 85 degrees of extension. This second strip of adhesive is carried around the ulnar aspect of the flexed forearm, from behind—forward, under the lower felt pad. The adhesive part extending from the forearm to the upper pad is covered with a third piece of adhesive. The second strip of adhesive is continued over the upper pad and down to the dorsum of the wrist joint. (Fig. 1.) In this manner, the adhesive plaster between the upper and lower pads is covered face to face in order to avoid any exposed adhesive surface. The patient is encouraged to be up and about the ward or to sit in a chair as much as possible.

The mechanics of this dressing is based upon the principle of a second degree lever. The fulcrum is located at the lower felt pad. (Fig. 4.) The downward force on the lever is composed of the weight of the forearm, hand and bag of weight. The upward force on the lever is exerted on the short arm just proximal to the lower felt pad. This upward force is carried to the humerus, which in turn, elevates the glenohumeral joint, thus raising the scapula and the acromion. The downward pull on the adhesive depresses the clavicle.

Simple sprains of the acromioclavicular ligaments are held in this form of "lever

dressing" until all symptoms subside. In our experience this has usually been necessary from four to fourteen days, depending upon the severity of the injury.

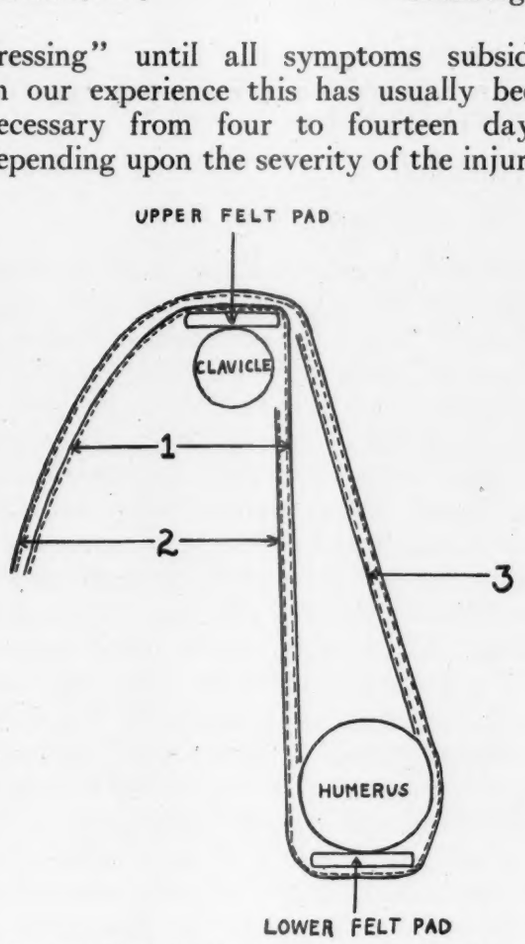

Fig. 3. Schematic drawing demonstrating how the three strips of adhesive are applied. The broken line demonstrates the adherent side of each strip.

When the case presented showed evidence of a mild subluxation, either clinically, roentgenologically, or both, the sling was kept intact for a period of three weeks. No stiffness of the shoulder or elbow joints was encountered after the sling was removed in any of these cases and all were returned to full military duty.

SUMMARY

Approximately fifty-three patients with mild to moderate injury to the acromioclavicular joint were treated with the "lever type" of sling described. Sixty-five per cent were simple sprains. Thirty-five per cent were diagnosed clinically and radiologically as mild subluxations. The sprains were immobilized for variable periods ranging from four to fourteen days, or until all tenderness subsided. The subluxations were immobilized for three weeks. No case was hospitalized more than four weeks. All patients were asymptomatic and had a full range of strong, active motion upon discharge to full military duty.

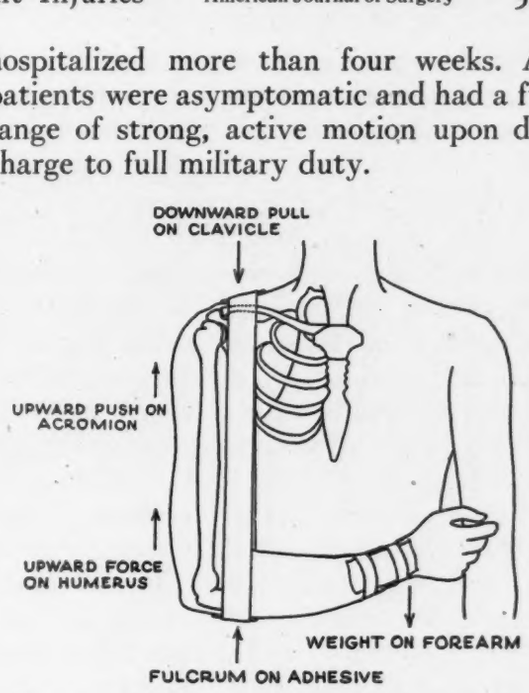

Fig. 4. A drawing to demonstrate the mechanical principles of the sling.

CONCLUSION

A modified "lever type" adhesive sling has been used in the conservative treatment of injuries to the acromioclavicular joint. The injuries varied from those of simple sprains to mild dislocations. All patients returned to full military duty. In the author's experience, this modified form of conservative treatment for the more usual injuries to the acromioclavicular joint has met with satisfactory results.

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MEAN DISPOSITION OF TIBIAL SHAFT FRACTURES

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SINCE it is well known that fractures of the tibial shaft are among that group of fractures more prone to non- and mal-union, an analysis of the statistical data of such fractures from the point of view of long-term results should be of interest.

Material. The cases reviewed were gathered from the clinical records at an Army General Hospital within the United States. In selecting the material, only cases that truly represented tibial shaft fractures were included. Thus Pott's fractures, tri-malleolar fractures, and fractures of the tibial plateau were excluded, as were fractures that mainly involved the femur and knee joint with minor involvement of the tibia, or fracture of the astragulus and os calcis with slight tibial malleolar involvement. These cases covered a two and a half year period and for the most part the patients were evacuees from the various combat theaters of the world. They include both simple and compound fractures, and these will be considered together for the sake of comparison. That more tibial fractures were treated is obvious, but an effort is made to consider only a selected group. This group numbers 120 cases, fifty-two (43 per cent) of which were compound, and sixty-eight (57 per cent) of which were simple. It is to be remembered that other fractures were present for the most part in these cases. The accompanying fractures ranged from fibular fractures of the same leg to concomitant fractures of the pelvis, patella, femur or bones of the ankle; but all cases fulfill the requirement of a tibial shaft fracture.

Initial Treatment. Initial therapy in most of the cases of simple fracture was the application of the Army leg splint and evacuation through the medical chain to a Field or Evacuation Hospital, where closed reduction was accomplished and casts applied. Many of these cases arrived with the second cast on, these being usually in excellent condition and the fractures in good position. Compound fractures usually received initial sulfa therapy locally and the Army leg splint was used. Débridement and open packing with vaseline gauze and application of a cast were usually carried out at the Field or Evacuation Hospitals. In more recent cases, courses of intramuscular penicillin have been used at times, effecting a seemingly somewhat earlier reduction in drainage. These cases usually arrived with their third or fourth cast intact and in good condition.

Analysis. Table 1 gives a breakdown of the cases from the point of view of the region of the shaft that was fractured.

TABLE 1

	Simple		Compound		Total	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
Upper third....	5	7	18	35	23	19
Middle third....	13	19	10	19	23	19
Lower third....	50	74	24	46	74	62
	68		52		120	

The more even distribution of the compound fractures along the shaft of the tibia is to be expected, for most of these occurred as a result of enemy action.

Other fractures were present in the group of sixty-eight simple fractures in sixty cases (88 per cent). Other fractures were present in the group of compound fractures in thirty-four cases (65 per cent) of the fifty-two cases.

Table 11 gives the mean disposition of

Table II

Disposition	Simple		Compound		Total	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
Duty	51	75	24	46	75	63
Discharged	6	9	16	31	22	18
Other	11	16	12	23	23	19
	68		52		120	

the cases thus far completed. Return to duty can be construed as a satisfactory result. The fact that a certificate of disability discharge was given, by no means precluded the possibility of the soldier returning to productive civilian life. The result "other" means for the most part that the soldier was transferred to another general hospital either nearer his home or for further treatment of some other condition, for example, peroneal nerve paralysis, of which four occurred in the whole group, one with a simple fracture and three with compound fractures.

Thus 75 per cent of simple, and 46 per cent of the compound fractures were returned to duty, with an average of 63 per cent of the cases being so terminated. In this total group of cases, twenty-six soldiers required operation, ranging from curettement to bone grafting. Ten of the operations were performed on simple fractures, three of these being returned to duty. Sixteen operations were performed on the group of compound fractures, ten of which ultimately returned to duty.

SUMMARY

The mean disposition of therapy in a group of compound and simple tibial shaft fractures has been presented with an average of 63 per cent of all patients being returned to duty.

THE VENOUS CIRCULATION IS AUDIBLE THROUGHOUT THE SYSTEM AND IN FIBROID UTERI

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SINCE publishing my article in 1922,[1] dealing with the fetal heart sounds, I have pursued ausculatory research in that field until I have hit upon the observation which forms the theme of this article.

Women have often told me that during their pregnancies they felt fetal movements at ten weeks of its duration. I followed this lead and I have heard so-called fetal heart sounds even at nine weeks of gestation with the only difference that they were of a much lower pitch than at four months or over; in fact I frequently had to strain my ears to hear them. I did not, for a time, venture to listen for them at an earlier period; if I did, I would have heard them earlier. The low pitch and apparent weakness of the sounds I attributed to the youthfulness of the fetus. Thus I continued for a number of years until I encountered a few abortions, miscarriages and premature births which I had occasion to examine a day or two previous to such interruption; and it was found that the fetus had either been absorbed already or it was macerated. It then dawned upon me that, in order to obviate further diagnostic errors, a bony medium might be a better conductor of sound and, if so, a better interpretation might be made through such auscultation I forthwith listened at, on, in front of and behind the anterior superior spine of the ilium even at a week or two of pregnancy and in non-pregnant women and in men and was awarded with the same results. There were low pitched sounds but partaking of all other qualities of the fetal heart. I concluded that since the arterial pulse can in most cases be heard synchronously, this can be nothing else but the venous pulse rate which is audible only instead of being palpable. Long before this observation I obtained identical findings in large fibroids of the uterus. That bruits resembling uterine souffles of pregnancy are occasionally heard in fibroid uteri is mentioned in text books. Two such cases have come to my notice. While I found no more uterine bruits, I heard in most cases a rapid, rhythmic sound of low pitch but sufficiently audible and having the characteristics of the fetal heart; that is, its rapidity was about twice the rate of the maternal heart with the irregularly alternating single and double beats of the fetal heart which latter I have amply written about in 1925.[2] At times the venous sounds are so low that one only experiences a consciousness of their presence and rate.

While the venous sounds, excepting those of the uterine fibroids may be heard at the least expected part of the body, it is best heard at the anterior superior spine of the ilium, the iliac fossa, the symphysis pubis, the upper part of the coccyx, the lumbar and sacral vertebra and over the long bones of the thighs and legs; and frequently over Pourpart's ligaments. In other words, they are best heard through the medium of bones or layers of muscles or both through which the veins cannot be readily compressed and the pulsations obliterated. Examples of muscle media instrumental in facilitating the audibility of the venous circulation are the muscles over (and including) Poupart's ligaments, fibroid uterus and pregnant uterus. The heart-like pulsations of the venous circulation is caused by the flapping of the venous valves in their encounter with the stream of circulating blood. It does not mean that the sounds

can be heard with equal clarity in all the areas mentioned, and in some of these areas they may at times be entirely inaudible.

In the beginning, before your ears are attuned to pick up these low pitched tones, select patients with normal or slow pulse rates; then count the pulse and impress on your mind its rate and rhythm. Listen for the venous circulation with the mouth of the stethoscope aimed at fitting snugly against the contour of the bones (not against the flesh) in order that there might be no leakage of sound. It may take one-fourth to one-half minute for the auditory transition to take place when the low pitched rapidity of the venous pulsations supersede the higher pitched, slower, arterial pulsations. Count. The venous pulsations will be more rapid and irregularly alternating single and double beats. If both pulsations are present, two or three of the venous beats will be heard between the arterial pulsations, depending upon whether they be single or double at that moment. If in doubt whether you are hearing the venous circulation or weak arterial pulsations, count the venous pulsations and compare with the radial pulse rate. The difference will be readily ascertained. At times the venous circulation is so distinct that it will help to make further elicitations comparatively easy. If not heard readily in an environment of quietude, concentrate your audition by pricking up your ears, closing your eyes and furrow your brow; and do not forget to eliminate friction sounds often caused by stethoscope.

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Case Reports

CAVERNOUS HEMANGIOMA OF THE SPLEEN*

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HEMANGIOMA of the spleen, a relatively rare condition, was reported for the first time by Virchow in 1846. Since then, little information has been contributed to the general knowledge of this condition. For a summary of the academic aspects of this subject, the reader is referred to the article by Pines and Rabinovitch.[8]

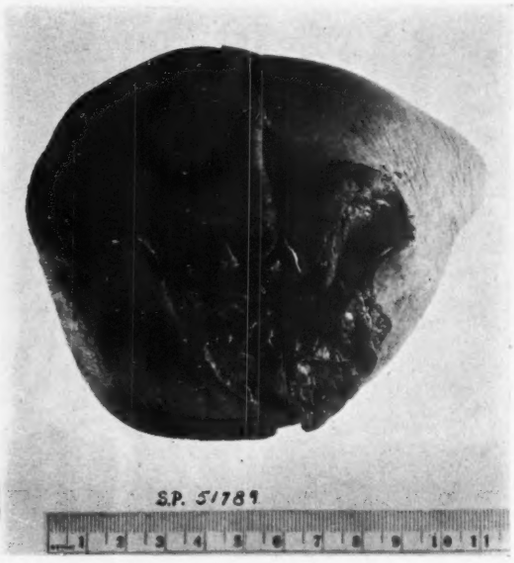

FIG. 1. Surface of spleen showing the rent on the diaphragmatic surface; S.P. 51789.

The pathogenesis of benign hemangioma is still undetermined. A developmental anomaly in the structure of certain vascular segments may be responsible for this lesion. Matas[6] and Delafield and Prudden[2] maintain that hemangioma is composed of the same cellular elements that give rise to primordial blood vessels. In accordance with this they believe that the hemangioma is the result of disordered, spontaneous growth of these primitive cells. Why hemangiomas assume a capillary or cavernous configuration and why they occur at all is imperfectly understood.

The incidence of benign hemangioma of the spleen is approximately .1 per cent. In 1927, Krumbhaar[5] reported one case in 6,500 autopsies. Kellert,[4] in 1932, reported three cases in 1,900 autopsies and considered this a high percentage. Schottenfeld and Wolfson[9] in 1937, cited four cases in 2,800 autopsies. In 1942, Pines and Rabinovitch[8] reported six cases of benign hemangioma of the spleen as incidental findings in 3,676 autopsies and stated that there was no clinical evidence of such a lesion. In a comprehensive review of the literature, Pines and Rabinovitch[8] reported forty-two cases of benign hemangioma of the spleen. Fifty per cent of these cases were found at operation and the other 50 per cent were incidental findings at autopsy. In 1943, Stuhlinger[10] reported a case of giant cavernous hemangioma of the spleen in which that organ weighed 4,700 Gm. The patient presented himself because of symptoms resulting from a large abdominal mass. After removal, 2 liters of bloody fluid were found in the hemangioma. In only three of the reported cases did preoperative rupture of the hemangioma necessitate splenectomy.

Haines and McIlroy,[3] in 1933, reported a case in a forty-three-year old white female who, with no history of trauma, complained of severe epigastric pain of twenty-four hours' duration, which was followed by nausea and vomiting. On

* From the Departments of Surgery and Pathology, School of Medicine, University of Maryland. Aided by a grant from the Syndey M. Cone Research Fund.

admission the patient was semicomatose and evidently in shock from blood loss. A tentative diagnosis of perforated gastric ulcer was made. Immediate operation was performed, and a spleen containing a ruptured hemangioma was removed. The patient died forty-eight hours postoperatively.

Fig. 2. Section of spleen showing the cavernous hemangioma and site rupture. S.P..51789.

In 1939, Nesler et al.[7] reported the first case of ruptured hemangioma of the spleen successfully treated by splenectomy. The patient was a white male, age forty, admitted with severe pain localized in the left side of the upper abdomen and referred to the left shoulder. The abdomen was rigid. The patient appeared acutely ill. The white blood cell count was 16,000. Physical findings suggested a perforated gastric ulcer. Operation was immediately performed. A ruptured hemangioma of the spleen was removed. The patient had an uneventful recovery except for superficial infection of the wound.

Cole and Forsee,[1] in 1940, reported a case in a twenty-seven-year old Army officer who had sudden intermittent pain in the upper abdomen associated with vomiting. The abdomen was distended and tender throughout. The white blood cell count was 15,200 and hemoglobin was 80 per cent. A tentative diagnosis of acute appendicitis was made. The abdomen was opened through a McBurney incision and blood welled into the wound. An appendicectomy was performed although that organ appeared normal. The McBurney incision was closed and an upper abdominal incision was made. Free blood was evacuated from the peritoneal cavity. A spleen containing a perforated hemangioma was removed. The postoperative course was uneventful.

The purpose of this paper is to add to the above reports a case of ruptured cavernous hemangioma of the spleen. It was mistakenly diagnosed as acute appendicitis and was successfully treated by splenectomy.

CASE REPORT

L. W., Chart No. 82835. A white boy, age sixteen, was admitted to the University Hospital because of generalized abdominal cramps of twenty-four hours' duration. There was no history of nausea or vomiting. Three weeks prior to admission he was hit by an automobile. He sustained abrasions of his left shoulder but this did not hinder him from working on a farm. The patient was first seen in the Accident Room with a temperature of 101°F., respirations 24, pulse 110, and blood pressure 120/80. The white blood cell count was 19,000. The urine was normal. Examination of the chest revealed dullness at the left base. The left diaphragm seemed fixed. *X*-ray of the chest revealed no positive findings. Severe pain and tenderness throughout the entire abdomen was noted but the tenderness was more marked in the left upper and right lower quadrants. Some rigidity with moderate muscle spasm was

present. A tentative diagnosis of acute appendicitis with early spreading peritonitis was made. Under spinal anesthesia the peritoneum was opened through a McBurney incision. The abdomen was found to contain a large amount of bright red blood. The McBurney incision was immediately closed and a left upper paramedian incision was made. Numerous small blood clots were seen floating in a pool of blood, approximately 750 cc. Upon palpation of the spleen, a large rent in the superior pole was found. (Fig. 1.) Several large blood clots were adherent to the left diaphragm. The free blood was evacuated. The liver, stomach and duodenum were found to be normal. Splenectomy was performed without drainage. The postoperative course was uneventful and the patient left the hospital sixteen days after operation.

The spleen weighed 450 Gm. and measured 16 by 11 by 7 cm. A large rent was present on the diaphragmatic surface. (Fig. 1.) Grossly, multiple hemorrhagic cysts were seen which occupied most of the splenic substance. They varied from 1 to 3 cm. in diameter. (Fig. 2.) The microscopic picture showed many large cystic vascular spaces filled with clotted blood. The cystic spaces had an endothelial lining and a definite fibrous wall. The splenic pulp was normal in appearance but displaced because of numerous angiomatous spaces.

SUMMARY

A benign hemangioma of the spleen sometimes ruptures causing hemoperitoneum and signs of an acute surgical condition of the abdomen.

Three of forty-three reported cases of hemangioma of the spleen were complicated by rupture and hemoperitoneum.

This condition, ruptured hemangioma of the spleen, has been most often diagnosed as acute appendicitis and perforated gastric ulcer.

A case of ruptured hemangioma of the spleen which was mistakenly diagnosed as acute appendicitis is reported. This patient was successfully treated by splenectomy.

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OBTURATOR HERNIA

REPORT OF AN OPERATION FOR IRREDUCIBLE INCARCERATION

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IN 1938, Watson collected 420 cases of reported obturator hernia since the first published observation of this uncommon entity by Arnaud de Ronsil in 1724. An analysis of this series showed that the hernia occurred with about equal frequency on the right and left side, and in twenty-one instances it was bilateral.

A personal review disclosed seven cases which appeared in the international literature in the past twenty years. None of these was accompanied by the complication of irreducibility which characterized the clinical findings in our patient.

Obturator hernia, as the name suggests, is an abnormal protrusion of an intra-abdominal viscus through the obturator foramen in the bony pelvis. This foramen transmits the obturator nerve and vessels, and is partially closed by a durable fascial membrane. Almost invariably it is a segment of small bowel that is found contained in the hernial sac, and this sac follows the course of the obturator nerve through the canal. In 1840, Howship described an important diagnostic sign which was later confirmed by Romberg (Howship-Romberg phenomenon). This consists of paresthesia and subjective pain from the hip downward to the knee along the antermedial aspect of the thigh. This corresponds to the anatomical distribution of the obturator nerve, and is apparently produced by pressure on the nerve from the sac and its contents in the obturator canal.

Obturator hernias occur most frequently in the thin, asthenic elderly women, who have borne many children. These conditions favor the absence of sufficient supporting fat in the retroperitoneal areas about the canal entrance. This together with attenuation of the fascial supporting structures in the pelvis, sets the stage. Precipitating factors are those which produce sudden increases in intra-abdominal pressure, i.e., coughing, sneezing or lifting.

Preoperatively, the existence of an obturator hernia may be suspected, but the diagnosis is seldom made before laparotomy. The clinical picture is one of small bowel obstruction, usually complete, with the absence of stools and flatus and with persistent vomiting. The Howship-Romberg sign was present in 16 per cent of 396 cases. Adequate study must be the rule with these patients particularly with regard to surgical preparation, for the mortality is high, 66 per cent in Watson's series.

Although two surgical routes, the abdominal and the obturator are theoretically available, the latter may be discarded in favor of a mid-line or lower rectus incision. An approach directly to the obturator foramen through an incision placed medial and inferior to the femoral canal is dangerous, and may require a supplementary laparotomy for proper exposure and successful disposition of the hernia.

CASE REPORT

Mrs. M. W. (No. 1362) a widow, age sixty-six years, was admitted to St. Elizabeth's Hospital at 1:55 P.M. on April 22nd with the complaint of severe pain in the left lower quadrant accompanied by vomiting and obstipation. A detailed history revealed that on April 18th, while attending an afternoon social gathering in the Church basement, she felt

vaguely sick and went home to bed. Two hours later she was seized with excruciating, intermittent pains in the lower left abdominal quadrant, radiating into the thigh. A few minutes later she vomited a large amount of bile-stained fluid with considerable force. The patient tolerated this condition for three days without relief, then agreed with the family that medical consultation was advisable.

On arrival at the hospital the patient appeared acutely ill. She was extremely dehydrated, the teeth were covered with sordes, and the tongue was dry and furry. Observation disclosed a thin distended abdomen with peristaltic waves sweeping from the ensiform to the pubes. A high-pitched tinkling sound was easily audible and on percussion a resonant tympany was elicited. Physical examination exonerated the usual superficial sites (femoral-inguinal-umbilical) for bowel incarceration. Rectal examination and subsequent barium enema furnished completely negative findings. On admission the temperature was 97.6°F., pulse 84, respirations 20, blood pressure 124/70. Blood was type O; Kline test was negative; there were 4.5 million red blood cells; hemoglobin was 97 per cent; white blood count was 18,900; there were 7 immatures and 11 lymphocytes. Five days later the red blood count was 5.1 million and the leucocytes had dropped to 7,500. Examination of the urine was normal.

Review of the family history furnished no pertinent information except to eliminate carcinoma as the cause of death in the patient's mother, father and ten siblings.

The patient stated that on December 26th, (four months prior) she experienced an attack of abdominal pain and vomiting similar to the present illness, but relatively less severe, which terminated spontaneously in twenty-four hours.

A continuous Wangensteen suction was started and fluids (5 per cent glucose with vitamin B in saline) were administered intravenously with very favorable effect on the distention and vomiting. After three days of preparation the patient was considered ready for surgery.

On April 26th, under local (0.5 per cent metycaine) anesthesia supplemented with intravenous pentothal (1.0 per cent), a lower right rectus incision was made through the thinned abdominal wall and the peritoneal cavity opened. There was no free fluid present. Due to the gastric suction, the intestines were not unduly distended and excellent visualization of the pelvis was obtained with the patient in the Trendelenburg position. The site of obstruction was easily discovered. A small loop of mid-ileum had become impacted in the left obturator canal. The bowel serosa appeared moderately injected and inspection revealed many broad sheets of tough fibrous tissue which intimately fused the involved intestine with the parietal peritoneum surrounding the obturator foramen. It appeared that this was a condition of rather long standing. Evidently the incarceration had suddenly become complete, transforming the partial obstruction into an acute emergency.

All attempts at reduction of the hernia by gentle traction were unsuccessful. Bimanual efforts, with pressure directed from outside the obturator canal also failed to release the bowel. Finding it impossible to effect reduction, it was decided to circumvent the obstruction. The afferent and efferent loops of bowel were identified and the serosa approximated by a continuous Lembert suture of fine cotton. A three-inch side-to-side entero-enterostomy was then completed. A No. 12 F. Pezzar catheter was placed in the small intestine distal to the anastamosis by a modified Stamm technic. This catheter was brought out through a separate stab incision, and was to function as a route for alimentary feedings during the convalescence. Two Gm. of sulfanilamide powder was dusted over the pelvic peritoneum and closure of the incision effected with interrupted sutures of No. 40 cotton.

The postoperative course was moderately stormy for seventy-two hours. Continuous gastric suction was required to control distention and opiates were administered to relieve the severe pelvic discomfort. On the fourth postoperative day, considerable flatus was passed and constituents of the enterostomy feedings (digested protein-dextrose-cod liver oil and soluble vitamins) were noted in the soft formed stool. Tests for urobilinogen in the feces at this time were strongly positive indicating a patent anastamosis. The incision healed by primary union and on May 1st (the fifth postoperative day) the gastric suction was removed and a soft diet administered orally. This was well tolerated by the patient

who was now pain free. On the following day the enterostomy feeding tube was withdrawn leaving a small fecal fistula which ceased draining forty-eight hours later. The patient became ambulant on May 5th and was discharged from the hospital in satisfactory condition on May 12th after a stay of twenty-one days. There has been complete bowel patency to date.

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Disruption or eventration of abdominal wounds usually occurs between the seventh and eleventh days after operation, after the use of vertical incisions closed with catgut. Wound infection, drainage, cough or other strain favors the disruption. After a secondary closure, a secondary eventration may occur.

From "Principles and Practice of Surgery" by W. Wayne Babcock (Lea & Febiger).

SPLENOSIS

INTRAPERITONEAL TRANSPLANTS FOLLOWING TRAUMATIC RUPTURE OF THE SPLEEN

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IN experimental animals splenic tissue may be readily transplanted. Von Stubenrauch[15] found on autopsy intraperitoneal transplants in dogs that had had crushed splenic pulp placed throughout the peritoneal cavity after splenectomy done three months previously. Marine and Manley[19] and later Perla[14] have successfully transplanted splenic tissue into the abdominal walls of white rats.

Were such experimental work done upon the human the results would be similar. We now know that under favorable conditions the human peritoneum has the ability to domicile as autografts fragments of splenic tissue that remain in the peritoneal cavity after traumatic rupture of the spleen. Hamrick and Bush,[5] in 1942, found in foreign literature only seven cases and in American literature three cases of multiple peritoneal transplants that had been discovered at celiotomy or at autopsy done months or years after splenectomy had been performed for traumatic splenic rupture. These, with a case of their own, make a total of eleven cases that have been reported. Buchbinder and Lipkoff[8] have suggested splenosis as a descriptive name for the condition. Gill,[7] in 1944, removed a mass of splenic tissue the size of a hen's egg from the left chest wall near the tenth rib of a negro man of fifty-two, four years after splenectomy had been done for a perforating gunshot wound of the spleen and of the diaphragm. Only diseased spleens rupture spontaneously and no case of autotransplantation has been reported following spontaneous rupture.

Von Stubenrauch,[15] in 1912, was the first to identify splenic nodules found over the peritoneum after traumatic rupture of the spleen as being autografts. Albrecht,[11] in 1896, Schilling,[19] in 1907, Von Kuttner[13] and Foltin,[12] in 1911, had reported having found similar intraperitoneal nodules which they had thought to be accessory spleens. Neither in Albrecht's case nor in Schilling's was there a history of trauma or splenectomy. To determine with certainty the origin of such nodules in patients who do not give the history of having had previous severe abdominal injury may be impossible. The number and the distribution of the nodules, however, may be suggestive. Multiplicity and wide dissemination with pelvic involvement from the effect of gravity characterize splenosis.

Accessory spleens, with rare exception, are few in number and are located within or about the splenic region in the left upper abdomen. They are miniature spleens of identical shape and structure as the parent organ with hilum, capsule, trabeculae, follicles and pulp. Grafts, on the other hand, vary in size and shape. They are often sessile but may be pedunculated. They have no hilum and blood vessels enter anywhere along the periphery. The parenchymatous structure is that of splenic pulp. Trabeculae are deficient and irregularly placed. Follicles occur only as they have survived as integral parts of the fragments of splenic tissue from which the grafts have sprung.

Accessory spleens are not rare. In a series of autopsy examinations done upon subjects of all age groups Adami and Nichols[17] found accessory spleens in 11 per cent. Jolly[16] found the autopsy incidence in children under seven to be 25 per cent and Sassuchin[18] in those under ten years to be 15 per cent. Congenital

anomalies are apt to be multiple and the increased autopsy incidence of accessory spleens in young children may be due to a greater mortality rate in them from associated developmental errors.

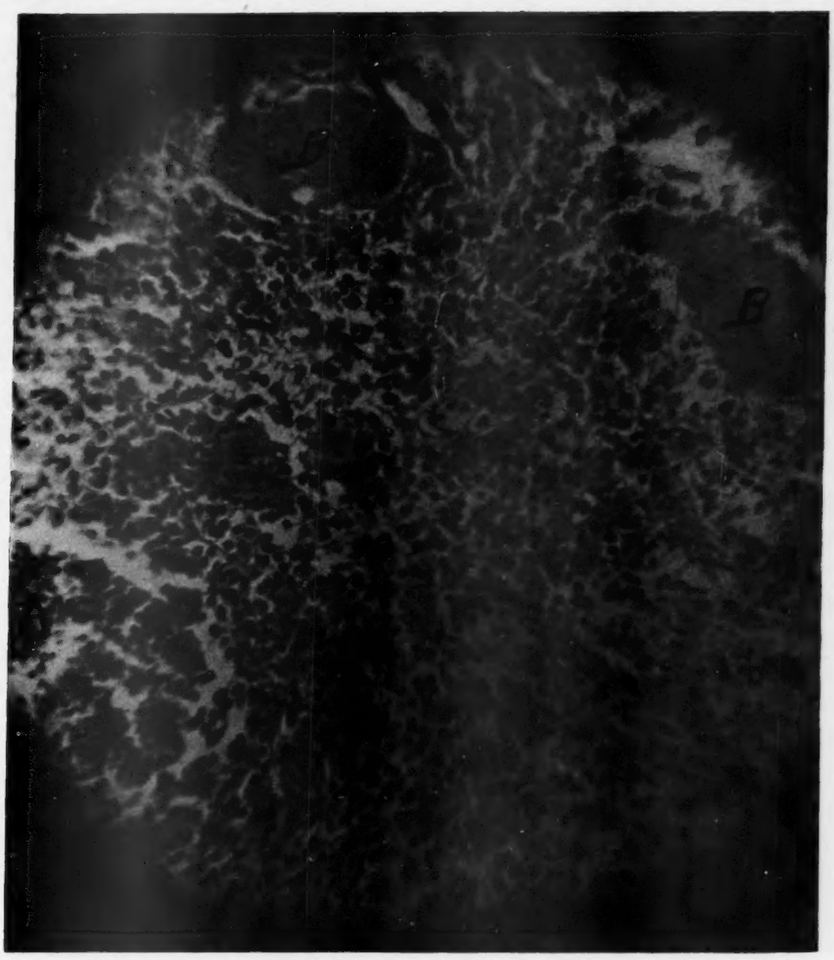

Fig. 1. Splenic transplant consisting of pulp with rudimentary follicle (A) and trabeculae (B), (low power).

Although well protected from trauma by position, the spleen is the most vulnerable of the abdominal viscera to the application of blunt force. The fact that it is a highly vascular lymphoid organ of reticulo endothelial structure with large communicating spaces makes it peculiarly susceptible to injury. A storage place for blood, it undergoes rhythmic contractions from the action of involuntary muscle fibers in the capsule and in the trabeculae. With the abdomen opened the blood volume just before splenectomy may be materially reduced by the administration of adrenalin. In the cat after death the spleen shrinks to one-fourth its original size.

Severe trauma may cause immediate rupture of the spleen with tearing of the capsule and exsanguinating intraperitoneal hemorrhage. Laceration of the friable splenic pulp from trauma of less degree, however, is followed by hemorrhage into the gland that may not cause rupture of the capsule from tension for a week or more after injury. Massive intraperitoneal hemorrhage may follow such delayed rupture of the capsule. Oneill and Rousseau[6] describe three types of bleeding occurring after traumatic injury to the spleen: (1) Extensive rupture with immediate massive hemorrhage; (2) small lacerations which ooze blood slowly over a period of hours or possibly days; and (3) delayed splenic rupture.

In the first and in the last of these classes at splenectomy, which is the only effective treatment, the diagnosis may be confirmed. In Class 2, however, the diagnosis remains uncertain for these patients are not subjected to operation. After abdominal injury pain referred to the left shoulder from irritation of the phrenic nerve may be from extravasated blood and suggests but is not pathognomonic of splenic rupture.

No one knows how many of us as children have had injuries to the spleen from trauma that did not require bed rest and was soon forgotten. Injury of this degree is probably more frequent than has been supposed. Spontaneous control of hemorrhage after rupture is possible. Babcock[10] states that hemorrhage into the abdominal cavity from injuries to the spleen may be partly, temporarily or permanently arrested by adhesion of coagula or omentum over the splenic wound (tamponade). If we accept the validity of this, we may no longer be sure that many of the so-called accessory spleens are not transplants. Although the question of origin is of academic rather than of practical interest, the burden of proof in doubtful cases may well be upon the advocates of accessory spleen.

In a review of 25,000 necropsies done at the University of Minnesota, J. S. McCartney found no case of healed splenic hematoma and Roettig et al.[1] of the Ohio State University say that such a spontaneous cure rarely if ever occurs. Since having become interested in a case of primary tuberculosis of the spleen with areas of calcification from healed tubercles we have seen a number of roentgenograms of non-tuberculous spleens in which similar shadows were thought to have been the remains of healed hemorrhagic infarcts. Infarction of the spleen is not rare and is described in text books. It is impossible to distinguish at autopsy a hematoma that has healed after trauma from one healed after infarction. A hematoma of either kind may heal with calcification.

It should be remembered that such nodules, whether congenital or acquired, possess the qualities of normally placed splenic tissue and that systemic disease resulting from splenic dysfunction may recur after splenectomy if the nodules are not surgically removed. In congenital hemolytic icterus and in thrombopenic purpura recurrence of symptoms after splenectomy are an indication for exploratory celiotomy in the attempt to find and to remove such nodules. Curtis[2] reports the case of a child of four having primary splenectomy successfully done by him during an attack of hemoclastic crisis. Return of clinical symptoms and of characteristic blood findings after four years were again followed by improvement after he removed two small accessory spleens at exploratory laparotomy.

CASE REPORT

R. B., age twelve, was admitted December 21, 1943, in an acute attack of recurrent appendicitis. At appendectomy done through a McBurney incision there were found perhaps a hundred dark bluish red parenchymatous nodules attached to the retroperitoneum, the mesentery and the great omentum. They were not pedunculated and were of irregular shape. Some were as large as acorns, others as small as peas. Several were found in the pelvis. The left abdomen and the splenic region could not be visualized. They were obviously glands of some kind, enlarged hemolymph glands, accessory spleens or autografts of splenic tissue. On section the tissue macroscopically was splenic pulp. The distribution was not that of accessory spleens. Following a fall three years previously, the boy had had splenectomy done by Dr. George Benet for traumatic rupture of the spleen with massive intraperitoneal hemorrhage. Convalescence had been uneventful and he had remained well except for recently recurring attacks of appendicitis. Since appendectomy he has been in good health.

Microscopical examination of a nodule removed by biopsy showed it consisted of splenic pulp with a peritoneal covering. This is without a doubt a case of autotransplantation following traumatic rupture of the spleen with wide dissemination throughout the abdomen of small fragments of splenic tissue by extrava-

sated blood. The condition in this patient was symptomless and of no clinical significance. We report this, the twelfth case of its kind, because of its rarity.

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Septicaemia and Bacteriaemia are due to the presence of organisms in the blood. In the former condition the organisms are not only present in the circulation, but actually proliferate therein.

From "A Short Practice of Surgery" by Hamilton Bailey and R. J. McNeill Love (H. K. Lewis & Co. Ltd.).

MEDIASTINAL EMPHYSEMA

REPORT OF THREE CASES SECONDARY TO NECK OPERATIONS

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THAT mediastinal emphysema should frequently follow surgical procedures on the neck is a logical conclusion if one considers the relation of the cervical fascial spaces to the mediastinum and the mechanics of breathing. A review of the literature does not support this conclusion. The purpose of this paper is to report three cases of mediastinal emphysema following neck operations and to suggest that the complication is much more frequent than reports indicate.

LITERATURE

Mediastinal emphysema has been recorded occasionally following thyroidectomy, more often following tracheotomy for laryngeal obstruction, and only rarely following all other procedures in the cervical region. Mediastinal emphysema was not mentioned as a complication of 21,869 thyroidectomies reviewed in reports by Dobson, Seely and Rose,[1] Adams,[2] Grace and Weeks,[3] Glenn,[4] Cochran,[5] McQuillan and Breidenbach,[6] Lahey,[7] and Spear.[8] Eighteen cases of mediastinal emphysema following thyroidectomy were reported in papers of Buford,[9] Gold,[10] Keis,[11] Barrie,[12] and Herman.[13] These included individual cases and seven cases collected from the literature. The rate of the complication was not stated in any of the reports. Fifty-two cases following tracheotomy for laryngeal obstruction were reported by Neffson.[14] Michaels,[15] Goldberg,[16] Work,[17] Schmidt.[18] and Barrie,[12] thirty of which complicated 126 tracheotomies for laryngeal obstruction in Goldberg's report. The three cases reported following other neck procedures include a tuberculous gland dissection (Buford[9]), a jugular vein ligation (Schmidt[18]), and a block dissection of the neck (Ackerman[19]).

COMMENT

Recent discussions of spontaneous and secondary mediastinal emphysema by Hamman[20] and by Schwartz, McIlroy, and Warren[21] stressed the spontaneous type. However, operations that open up the fascial spaces of the neck with their direct communication to the mediastinum offer an ideal condition for the force of inspiration to suck air into the mediastinum. Any struggle during anesthesia increases this force. Any obstruction to the normal air passages transfers the effective force of inspiration from the normal air passages to the opening into the mediastinum made by the surgeon.[17]

The presence of mediastinal emphysema is detected by finding over the precordium bubbling crepitations synchronous with each heartbeat, which sounds may be present in only certain positions of the patient. When once heard they are confused with no other sound. Pain similar to the pain of angina may be present or absent. Roentgenograms at times disclose the gas around the heart shadow and often pneumothorax, which is a common accompaniment. In severe cases there is respiratory difficulty and signs of cardiac tamponade.[20]

Asymptomatic cases require no treatment. Rest, reassurance, and analgesics suffice in mild cases.[22] Oxygen is indicated in cases with respiratory difficulty.[9] Patients with severe cases need the air evacuated from the mediastinum, which evacuation has been accomplished by aspiration[23,24] or by incising the base of the neck to allow the trapped air to escape.[25,26] One patient in extremis was saved by the heroic measure of splitting the sternum.[20] The free airway which intratracheal anesthesia affords is good prophylaxis.[27] Local

anesthesia obliviates any struggle from anesthetic induction or faulty anesthetic technic. In operating upon the neck it is well to keep moist gauze in the lower end of the wound to seal off the neck spaces from the mediastinum.[19]

Whereas the cases reported previously have been severe, often resulting in death, the cases in the present report have been mild.

CASE REPORTS

Case I. A twenty-seven year-old white male was admitted March 30, 1943, for dizziness, precordial pain, and palpitation present since September, 1942. Physical examination showed an enlarged right lobe of the thyroid with no roentgenologic evidence of a substernal mass. There was no evidence of toxicity. On April 9, 1942, the right lobe of the thyroid was removed under inhalation anesthesia. The induction was smooth and at no time was there respiratory distress or cyanosis. Difficulty was encountered, however, in freeing the gland posteriorally and from the trachea. All bleeding was controlled and the wound closed without drainage. The patient tolerated the operation with very little change in pulse or blood pressure. Several hours after his return to the ward the patient complained bitterly of severe precordial pain with radiation down the left arm. Physical examination was normal except for precordial bubbling crepitations synchronous with each heartbeat. Roentgenogram showed subcutaneous emphysema in the neck and mediastinal emphysema. There was no pneumothorax. Morphine subcutaneously and reassurance relieved the symptoms in a few minutes. The remainder of the convalescence was uneventful. No signs of mediastinal emphysema could be found after the third postoperative day.

Note. Nineteen thyroidectomies have been done in this hospital since February, 1943, and in only the above case was any evidence of mediastinal emphysema found. The diagnosis was quite important in this case, especially since the patient came to the hospital complaining of precordial distress.

Case II. A thirty-two year-old white male was admitted October 17, 1943, with a self-inflicted knife wound of the anterior neck penetrating the thyroid cartilage. A tracheotomy tube having been placed in the incised trachea and bleeding having been controlled by suture of the wound by the local medical officer, the condition on admission was excellent with no respiratory distress and no shock. Blood pressure was 160/100; pulse was strong and slow; the heart and lungs were normal. Under cervical plexus block and local anesthesia the wound was thoroughly cleansed, bleeding controlled, tracheotomy done, and the wound repaired with cotton sutures. The patient was quiet and in good condition throughout the procedure. The night after admission the tracheotomy tube, which was too short for the patient, came out of the trachea. The next day subcutaneous emphysema was found around the tracheotomy opening, and bubbling crepitations synchronous with each heartbeat were heard over the precordium. A larger tracheotomy tube was inserted under local anesthesia. By the fourth postoperative day there was no evidence of mediastinal emphysema. The patient's further convalescence was uneventful, the tracheotomy tube being removed on the sixth postoperative day.

Note. The asymptomatic mildness of the mediastinal emphysema called for no treatment except insertion of a larger tracheotomy tube.

Case III. A twenty-seven year-old white male was admitted on April 23, 1945, following an explosion in which he received multiple wounds. The most severe laceration was the one in the left supraclavicular region. After shock treatment and under cervical plexus block the wound was enlarged and the clavicular head of the sternocleidomastoid muscle was transected. The internal jugular vein was dissected free, and a piece of jagged metal about an inch across was found lodged in the base of the neck. Bleeding was controlled by fingers placed over the internal jugular vein at its junction with the subclavin. On releasing the finger the wound became filled with blood and milky white chyle. This was evidence that the main lesion was behind the clavicle, so that inhalation anesthesia was started. During induction the patient strained and coughed and became cyanotic, having evidently developed a laryngeal spasm. Sucking sounds were heard around the vessels of the neck when the

patient attempted to inspire against the closed glottis. To facilitate the exposure, the clavicle was severed with a Gigli saw and the medial fragment mobilized. There was a large tear about an inch long at the junction of the internal jugular and subclavian veins on the lateral aspect. The thoracic duct was visualized and appeared uninjured. The vein was repaired with cotton sutures and the wound closed in layers after wiring the clavicle. Immediately after the operation bubbling crepitations synchronous with each heartbeat were heard over the precordium. The patient was heparinized and given sulfadiazine and penicillin. He had a rather stormy postoperative course, developing a cloudy effusion of his left chest which was aspirated and penicillin injected. This fluid was negative for fat. The third day after the accident no signs of mediastinal emphysema were found. The patient's symptoms and signs all subsided and he became well. His fractured clavicle was treated by a T-splint. On June 18th he had remained symptom-free for several weeks and was being kept in the hospital for his fractured clavicle. At no time did his mediastinal emphysema give him any distress. He never developed evidence of pneumothorax.

Note. As this mediastinal emphysema was asymptomatic and mild, no treatment was required for this condition. During the induction of the anesthesia when the patient strained with his internal jugular vein dissected out, the remark was made that mediastinal emphysema would probably develop.

SUMMARY

1. In two years three cases of mediastinal emphysema following neck operations have been found in one air force regional station hospital where these operations are relatively infrequent. This suggests that the complication is more prevalent than previous reports indicate.

2. The cases were all mild, which is also a marked contrast to cases in other reports.

3. Routine auscultation of the precordium following surgical procedures on the neck will surely bring to light many more incidents of this entity.

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To increase exposure, especially in the upper abdomen, a transverse incision may be made through the skin and anterior and posterior sheaths of the rectus muscle at right angles to a median incision. This type of exposure may be indicated in certain operations upon the gall-bladder, stomach, pancreas, spleen, and transverse colon.

From "Operations of General Surgery" by Thomas G. Orr (W. B. Saunders Company).

VOLVULUS OF THE SIGMOID COLON*

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VOLVULUS of the sigmoid is an infrequent but often spectacular cause of acute or intermittent intestinal obstruction. Sweet[1] found that fifty-three of 540 cases of acute intestinal obstruction were due to volvulus and of these, thirty-six involved the small intestine, ten the sigmoid, and six the cecum. In Central European countries[2] the relative incidence is much greater than in America; in Russia volvulus has been reported in as high as fifty per cent of acute intestinal obstructions. This greater incidence has been explained by the more bulky vegetable diets of these peoples.

The present case is reported because of several unusual features.

CASE REPORT

The patient was a twenty-year old soldier of Polish and Hungarian parentage. The diet had not been unusual in quantity or type of food, and the bowel habits were regular without the use of laxatives.

The earliest symptoms were noted about a year prior to the present admission when there was an attack of intermittent, cramp-like, low back pain which lasted several hours. Abdominal symptoms were not present and there was no radiation of the pain. In the year following there were six or seven such attacks, some of which were associated with abdominal cramps, and most of which were accompanied by a painful desire to defecate. On January 29, 1945, there was an episode of low abdominal cramps which again were associated with a desire to defecate. This was relieved by magnesium sulfate and the passage of a well formed stool. The bowel movements during the next ten days were frequent but consisted only of small amounts of yellow mucus and some flatus. On February 9, 1945, he was awakened by severe intermittent pain in the low back, localized over the sacrum. This pain did not radiate to the abdomen or elsewhere, but there was again the urge to defecate and vomiting of a small amount of greenish material.

On admission the patient was obviously in marked distress. Examination of the back revealed nothing abnormal, there was no objective tenderness or muscle spasm. The abdomen was full but not distended or tense. Abdominal tenderness and muscle resistance were completely absent. No masses were palpable and peristaltic sounds were of normal intensity. Rectal examination revealed no tenderness but a peculiar soft wall of resistance was encountered superiorly. Sigmoidoscopic examination for a distance of 25 cm. revealed an empty bowel of normal appearance. The leucocytes numbered 14,750 with 84 per cent neutrophiles and 11 per cent lymphocytes; the temperature was 99°F. Roentgenograms of the abdomen (Fig. 1) revealed a tremendously dilated loop of large bowel which occupied most of the mid-abdomen: the septa were greatly exaggerated. The remaining large bowel, including the cecum, was not distended and the small intestine was not visualized. In the erect position (Fig. 2) a fluid level was evident within the dilated loop. Volvulus was suspected and its location in the sigmoid was demonstrated by a small barium enema. (Fig. 3.)

Immediate exploration was performed through a left rectus incision. The greatly dilated loop of sigmoid presented in the wound. It filled the entire left and part of the right abdomen and possessed a wide, fan-shaped portion of mesentery 11.5 cm. in length and 6.5 cm. across its base. Torsion of 360 degrees had resulted in almost complete obstruction at both the proximal and distal ends of the loop. The bowel was moderately dusky in

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color but viable; it was deflated through a previously inserted rectal tube, then delivered and rotated in a clockwise direction to restore its normal relationship. A Rankin obstructive resection of the redundant loop was performed. The convalescence was uneventful except for atelectasis of the right lung which became apparent on the fourth day and promptly responded to postural drainage and medical management. The colostomy was closed May 22, 1945.

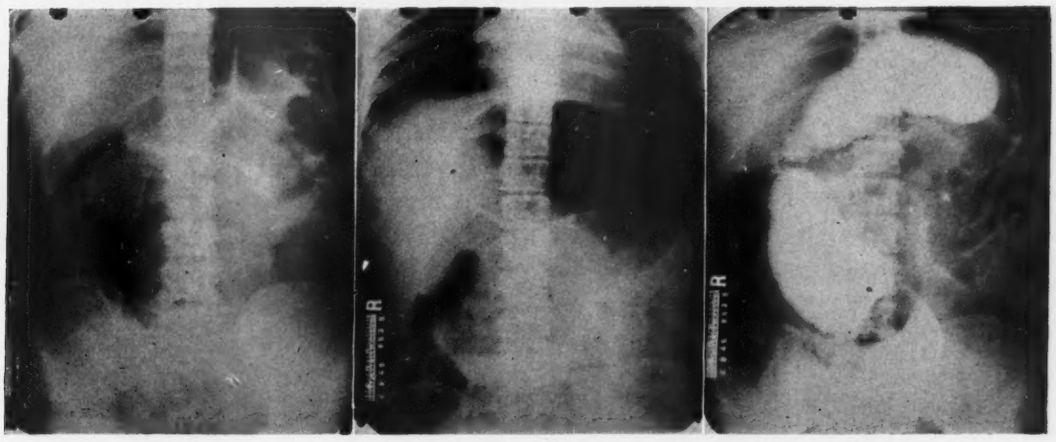

Fig. 1. Fig. 2. Fig. 3.

Fig. 1. Roentgenogram of volvulus of the sigmoid showing tremendously dilated loop of colon.

Fig. 2. Volvulus of the sigmoid in erect position, a fluid level is apparent in a dilated pelvic loop of colon.

Fig. 3. Roentgenologic appearance pathognomonic of volvulus of sigmoid, the barium enema reveals rapid tapering of the rectum to a constriction with torsion of rugal folds with a tremendously dilated loop of sigmoid above it.

The specimen (Fig. 4) consisted of a hugely dilated segment of sigmoid 15 cm. in diameter and 71 cm. along its anti-mesenteric border. It was attached to a widened, fan-shaped segment of mesentery. No intrinsic obstruction was found. The mucosal folds were obliterated, the bowel wall greatly thinned, and large septa were present.

COMMENT

This case is of interest because of the presenting complaint of backache without abdominal pain, tenderness or distention, The ineffectual urge to defecate was the most significant and only localizing symptom, and it suggested a diagnosis of high prolapse of the rectum, pedunculated tumor of the rectosigmoid, or volvulus of the sigmoid colon. Roentgenograms were typical of volvulus of the sigmoid colon. Volvulus produces a closed loop type of obstruction and interference with the mesenteric blood supply, hence distention of the isolated loop is usually rapid. This contrasts with the more general distention of the proximal colon and cecum characteristic of simple obstruction of the sigmoid such as results from malignant lesions. Roentgen studies with the barium enema demonstrated a twisting spiral pattern in the rugal folds with rapid filling of the dilated loop above the obstruction. In this instance reduction of the volvulus did not occur during examination as sometimes happens. The interference with the

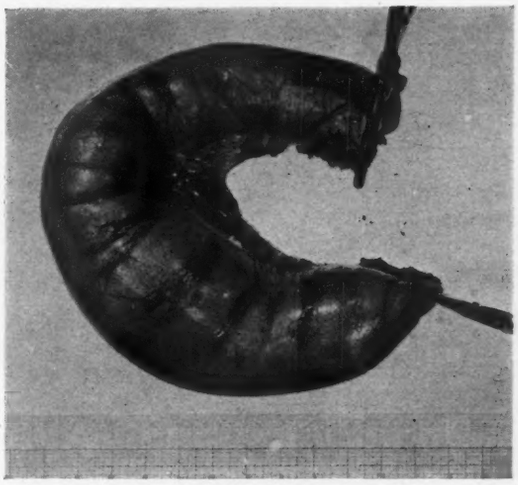

Fig. 4. Specimen removed from volvulus of sigmoid; megacolon with widened mesentery.

circulation resulted early in a relatively quiet abdomen. The history of this patient suggests that episodes of partial or temporary obstruction had occurred at intervals for a year before complete obstruction led to surgery. The localized redundancy of the sigmoid mesentery was an essential prerequisite to the production of the volvulus and probably was a congenital anomaly. In addition to the dilatation there was also actual hypertrophy of the involved segment of colon, which was either congenital in origin or secondary to the repeated episodes of chronic intermittent obstruction, or the result of both of these factors.

When volvulus is diagnosed immediate surgical intervention is indicated. Preoperative preparation other than essential intravenous fluids only results in unjustified delay which often permits the development of gangrene and peritonitis. Attempted deflation of the distended loop by intubation is futile since the obstruction is of the closed loop type. When strangulation and gangrene are demonstrated at exploration, removal of the damaged gut is imperative. Early, before extensive vascular damage has developed, simple untwisting of the volvulus will not prevent recurrences because the redundant loop with its long narrow pedicle of mesentery remains uncorrected. The Rankin obstructive resection is admirably suited to any of these situations because it is easily and quickly performed and is attended by a reasonably low mortality even in seriously ill patients.

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RUPTURE OF THE DEEP EPIGASTRIC VESSELS

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SO-CALLED "spontaneous" rupture of the deep epigastric vessels, while not a common accident, is such a dramatic entity and is of sufficient diagnostic importance that it must always be borne in mind. This is cause sufficient to warrant the report of two cases. Observations and deductions based on two cases cannot be conclusive, but are so opposed to the trite handed-down concepts, as to justify a modern rationalization of the etiology and basis of symptoms.

CASE REPORTS

CASE I. No. 17101, G. T., age forty, a white female, was admitted to St. Joseph's Hospital, April 20, 1938, at 3 A.M., with a chief complaint of severe right-sided abdominal pain. Four hours previous to admission while taking her usual reducing exercises of bending over and touching the floor, she was suddenly seized with severe, agonizing pain. It was not accompanied by nausea or vomiting.

She had had the usual diseases of childhood. She had had two previous abdominal sections, an appendectomy in 1919, and a cholecystectomy in 1929.

Physical examination disclosed a stout, middle aged woman rolling around in bed obviously in severe pain. Examination was essentially negative except for the abdomen, which was tender throughout, much more marked on the right side. There was board-like rigidity of the right side, and very marked splinting of the left side. The white blood count was 16,100 and the red blood count was 4,100,000. The temperature was normal.

Preoperative diagnosis: Pelvic inflammatory disease with intestinal obstruction.

At operation a lower right rectus incision was made. On splitting the rectus muscle, large clots were evacuated from the hematoma coming from the ruptured epigastric vessels. Both the artery and vein were torn. The clots were evacuated the bleeding points ligated. The peritoneum was opened for exploration. The viscera were normal. Adhesions from previous operations were encountered and separated. The wound was closed in layers. One piece of iodoform gauze was used for drainage. Convalescence was uneventful and the patient was discharged from the hospital on the tenth postoperative day.

CASE II. No. 35896. M. D., age 59, a white female, was admitted to St. Joseph's Hospital, January 16, 1943. Her chief complaint was pain in the lower left quadrant. On the day of admission following a severe paroxysm of coughing she suddenly developed severe pain in the lower left quadrant. The pain became excruciating but was not accompanied by vomiting. Her symptoms became progressively worse, accompanied by shock. She was admitted to the hospital apparently acutely ill.

She had had no previous operations and remembered no serious illness.

Physical examination revealed the patient to be a thin emaciated adult, acutely ill, in severe pain. Examination was essentially negative except for the abdomen. There was marked rigidity and exquisite tenderness of the entire left side. There was a palpable mass, the size of a large grapefruit, in the lower left quadrant. Her white blood count was 13,400 and red blood count 3,500,000. Her temperature was normal.

Preoperative diagnosis: Carcinoma of the colon with perforation.

Under spinal anesthesia the abdomen was opened by a lower rectus incision. On opening the sheath of the rectus muscle a large hematoma was encountered coming from the deep epigastric vessels. Both the artery and vein had been torn apart. The hematoma had dissected its way beneath the symphysis pubis. The vessels were ligated and the clots evacuated. The peritoneum was opened and the exploration was negative. The wound was closed in layers with one small piece of packing for drainage.

"Spontaneous rupture," "epigastric apoplexy," and "idiopathic rupture" are

misnomers. There is a definite cause if careful attention is given to the history (Case I—exercising; Case II—coughing).

Incidence. No statistics based on a given number of hospital admissions was found in the literature. Richardson reported the first case in this country in 1857. Payne states that from 1928 to 1938, 165 cases have been reported and he estimates that twice that number were not reported. Therefore, while not common, it could not be called a rare disease.

Anatomy. Max Brödel has made a masterful contribution on the anatomy. The rectus muscle with its long narrow belly is really made up of separate muscles that act as one, because of the tendinous bands that run transversely. It can contract and expand as much as nine-finger breadths. Terrific pressure is developed when the body is raised from the horizontal to the sitting posture (Case I) and in coughing or the act of defecation (Case II). Coursing beneath this muscle from the femoral artery below, and the internal mammary artery above, this longest anastomosis in the body is fed by large caliber vessels with a relatively high pressure. The vessels lie free in loose areolar tissue separated from the abdominal cavity by peritoneum and thin fascia. They are attached, however, to the muscle by branches and are closely adherent at tendinous inscriptions.

Pathology. The rupture of the epigastric vessels usually occurs low down in the sheath below the fold of Douglas (both cases). The older descriptions divide it into an "acute" or "chronic" affair depending upon the rapidity of the progress. They speak of arterial bleeding and of venous source. In my two patients both the artery and vein were torn asunder with rapid massive hemorrhage. It is easily conceivable that with the same etiology lesser tears could occur with injury to only the vein or artery and the symptoms would be directly proportional to the amount and extent of the extravasated blood.

Etiology. In both instances there was alternate relaxation and violent contraction of the rectus muscle with a rapid and relatively enormous change in the size of that muscle. The vessels tightly bound and an integral part of the muscle at intervals, and loose in between, simply are unable to accommodate to the changing condition and were torn apart by the crack-of-the-whip action. The old accounts still speak of atheromatous vessels and debiliated disease such as typhoid. This of course, I cannot subscribe to. The frequent reports of occurrence in pregnancy and whooping cough would, however, lend weight to my conception. Likewise, the frequent occurrence of "right rectus strain" in boys training at parachute school bears out the etiological factor.

Symptomatology and Physical Signs. The onset is sudden with severe pain and exquisite tenderness in the abdomen. While previous descriptions all mention nausea and vomiting as symptoms, they were conspicuous by their absence in both cases, and that factor alone should have been a warning against an intraperioneal lesion. Prostration and shock are prominent and pronounced. The leukocyte count was moderately increased; fever was not present.

A palpable mass may be present (Case II) limited to the rectus muscle, but muscle guarding and rigidity may make it impossible to palpate until the patient is under anesthesia (Case I). Ecchymosis of the abdomen is reported having been present but did not occur in the patients observed, and would require an injury to the anterior sheath of the rectus which is most unlikely. I consider it unreliable.

Forthergill's sign depends on being able to feel a palpable mass which is still present when the patient sits up, and this mass cannot be moved from side to side. While this would be relatively conclusive evidence, it would be very difficult to elicit as an early symptom.

Diagnosis. I failed to diagnose both cases. In retrospection this was inexcusable.

The differential diagnosis is easy. A patient seized with severe one-sided abdominal pain following exertion that places strain on the rectus muscle, accompanied by shock, marked rigidity and exquisite tenderness limited to one side, not accompanied by nausea or vomiting, with or without a palpable mass will forever bring to my mind the possibility of ruptured epigastric vessels. Once the possibility is considered there is little likelihood of error and sufficient has been accomplished.

Treatment. Surgery offers the only treatment: incision, evacuation of the clots, ligation of the vessels and placing of a small drain. The peritoneal cavity need not be opened, but there is no contradiction to so doing if one wishes to explore.

CONCLUSION

Two cases of rupture of the deep epigastric vessels are reported. The injury is not "spontaneous" but follows a definite muscular action. This fact could be important from a medicolegal or industrial standpoint. A logical theory based on atomical and mechanical facts is presented to explain why the deep epigastric vessels are subject to rupture. In civilian life, the preoperative diagnosis has seldom been made. The cardinal signs and symptoms have been presented. If the entity is borne in mind, careful observation and logical deductions will readily make the diagnosis.

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DESMOID OF THE ANTERIOR CHEST WALL

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DESMOIDS or fibromas are infrequently occurring tumors of the musculofascial structures usually appearing on the anterior abdominal wall. Their presence at other sites has been noted although their appearance elsewhere than upon the abdominal wall is distinctly in the minority. Pearman and Mayo have reviewed the literature rather carefully and have reported the details of seventy-seven patients with desmoids who were treated at the Mayo Clinic. In twenty-two instances in their series, the tumor was located in the skeletal muscle system removed from the anterior abdominal wall. The incidence in females in these twenty-two cases was greater than that among males, as is usual with this particular tumor at any site.

The casual factors underlying the appearance of these tumors are unknown. However, a history of trauma is frequently obtained and was noted in 60 per cent of the seventy-seven cases reported by Pearman and Mayo.

The pathologic characteristics of these tumors are somewhat reminiscent of a leiomyoma of the uterus. The structure is tough, grates under the knife and the cut surface bulges. The interlacing bundles are clearly visual to gross inspection. The histologic structure is that of a rather cellular fibroma which is not encapsulated. The surrounding and infiltrated muscle fibers are attenuated and foreign body-like giant cells of muscular origin are visible.

The tumors do not undergo metastasis. They may, however, recur locally if incompletely removed.

The patient whose case report follows is of interest because he is a male, a definite history of trauma was present, and the tumor was found in the upper anterior portion of the chest wall intimately connected with the pectoralis major muscle.

CASE REPORT

The patient is a twenty-three year old white male who incurred a wound of the right thorax from a shell fragment. He suffered a fracture of the right humerus, a laceration of the right lung and a fracture of the right coracoid process. Several thoracenteses were done and later a thoracotomy with removal of the foreign body was performed. Two months after injury, the patient noted a small mass in the right infraclavicular region. Gradual growth was noted. The mass was tender and pain at the site of the tumor was noted upon exercise.

Physical examination was not noteworthy with the exception of the presence of the tumor. A firm, rounded mass measuring about 5 by 3 cm. was found in the right infraclavicular region over the first rib. (Fig. 1.) The skin was freely movable over the tumor which was firmly attached to the deeper structures. Other tumors were not found during the examination.

Various laboratory procedures including determinations of the blood calcium, phosphorus and phosphotase were done as we felt a tumor of bony origin might be present. These determinations were all found to be within normal limits. Likewise the roentgenograms of the chest and the skeleton did not reveal a bone tumor or tumors to be present. We were thus confronted with a firm tumor apparently not of osseous origin but which was firmly attached to the deep structures in the right infraclavicular area.

Photographs of the patient using the infrared technic did not show any dilated venous channels about the tumor. Some idea as to the depth to which the mass extended was obtained from a phlebogram of the right upper extremity. Twenty-five cc. of diodrast were injected into a vein in the lower right forearm with a tourniquet about the mid-arm. A pressure of 30 mm. of mercury was maintained during the injection. A visualization of the right brachial, subclavian and innominate veins was obtained

and no perceptible deviation from the normal in size or position of these blood vessels was noted.

Fig. 1. Right infraclavicular tumor.

Surgical exploration was the next procedure as all attempts to establish a diagnosis were without avail. A curvilinear incision over the tumor was made. The mass was of firm fibrous consistency without definite encapsulation, infiltrating the pectoralis major muscle and firmly attached to the fibrous tissue over the sternoclavicular joint. A wide block resection of the tumor and adjacent muscle was done.

The postoperative period was without incident. The wound healed quickly and firmly.

The tumor was 5 cm. in diameter and upon cut section presented a mass of coarsely reticulated fibrous tissue flecked with yellowish areas. (Fig. 2.) The peripheral portions of the tumor were hemorrhagic and stained yellowish-brown.

Microscopically, the specimen consisted of a mass of intermingled broad connective tissue fibers among which were numerous capillaries and foreign body giant cells. Infiltration of the

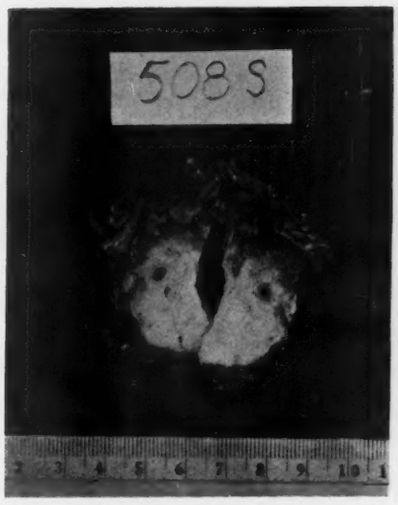

Fig. 2. Cross-section of the tumor.

surrounding muscle by the connective tissues fibers was noted. Numerous areas of hemorrhage and foreign body giant cells were seen at the periphery of the lesion. The intermingling of the fibrous core, the giant cell reaction and the apparent infiltrative character of the tumor classified it as a desmoid.

SUMMARY

The tumor is of interest for several reasons. It occurs in males but in a minority of cases. Likewise it usually is found in connection with the musculo-aponeurotic structures of the anterior abdominal wall and only occasionally in other sites. A definite history of trauma was obtained from the patient which gives further support to the theory of traumatic origin in connection with individual predisposition as a cause for the appearance of these tumors.

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OSTEOID OSTEOMA

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OSTEOID osteoma is a relatively new entity, the original article by Jaffe[1] first describing this condition appearing in 1935. Since then a later article by Jaffe and Lichtenstein,[2] in 1940, and another by Lewis,[3] in 1944, have come to the attention of the authors.

Jaffe and Lichtenstein report thirty-three cases, with rather wide skeletal distribution, exclusive of ribs, innominate bones and skull. Lewis, in his series of eleven cases, nine proved and two which he believes fall into this classification, reports one case of proved osteoid osteoma occurring in an innominate bone (right ilium).

The lesion is a small benign slowly growing osteogenic tumor, composed of osteoid tissue which later becomes calcified and converted into hypercalcified atypical bone. This nidus characteristically produces an excessive bone reaction about it so that, as it progresses, this sclerotic new bone formation may obscure the nidus on the roentgenogram. The true nature of the condition can be brought out by overexposing the film as was done in our case.

The usual history is that of localized pain, mild at the start, worse at night, which becomes increasingly severe and may in time interfere with sleep. Local swelling may also be noticed. Limp, stiffness and weakness may also be present. A history of antecedent trauma is not common.

Findings on examination consist of local tenderness, swelling, limp and sometimes muscle atrophy. Local heat, redness and fever are absent. In our case there was a very definite tendency for the patient to protect the affected part against even slight trauma.

A differential diagnosis must be made between bone abscess and chronic osteomyelitis, the most frequent diagnosis being "chronic sclerosing non-suppurative osteomyelitis." It has also been diagnosed osteoma, syphilitic osteoperiostitis and sclerosing osteogenic sarcoma.

We believe that while the diagnosis is usually made on the roentgenogram the condition should be suspected clinically when the patient is a young adult presenting localized bone pain of some months' duration. There may or may not be a palpable tumor mass of bony consistency. The lack of local heat or redness and of systemic findings of infection as demonstrated by fever, abnormal blood count and increased sedimentation rate, should bring this condition to mind.

We present the following case with characteristic roentgenogram, as we believe the condition is not commonly recognized and because this benign tumor is most frequently seen in the type of young adult with whom we in the army have to deal.

CASE REPORT

A white male, age twenty-seven, was admitted to this hospital, November 7, 1944, complaining of pain in the right forearm. His history was that in February, 1943, after a very minor injury to his right forearm for which he did not seek medical advice, he developed occasional attacks of local pain which would occur about twice a month. This pain could be induced by relatively minor local injury such as hitting his arm against an object. In October, 1943, at a Station Hospital in the States an *x*-ray was taken. The patient was told he had a small bone tumor. No surgery was recommended and he was allowed to proceed overseas. Since that time the patient stated the local pain had become more frequent and more severe and occasionally interfered with sleep. He unconsciously found himself

guarding his arm against contact with objects. He had noticed swelling over the forearm, and tenderness on pressure but no local heat. He

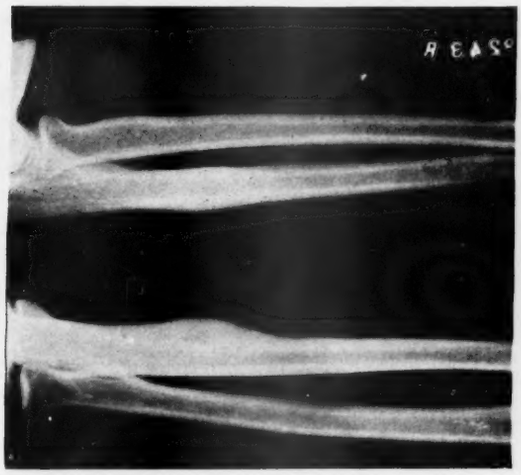

Fig. 1. Preoperative picture showing osteoma of ulna with nidus in center.

did not remember having any unexplained temperature rises.

Examination showed a hard bony tumor mass, which appeared to arise from the middle third of the right ulna. This was exquisitely tender on pressure. There was no local heat, redness or muscle atrophy present. Laboratory findings were completely negative and there was no fever.

A roentgenogram (Fig. 1) taken November 18, 1944, showed an area of bone production and sclerosis surrounding a translucent area in the cortex of the ulna on the posteromedial aspect. This was diagnosed an osteoid osteoma.

Operation was performed on November 25, 1944, at which time the tumor was removed along with cortical bone down to the medullary cavity. The nidus was not identified as such. All removed bone fragments were sent to a neighboring laboratory for examination, as our hospital is not equipped to do bone pathological examinations. Unfortunately only two of the many bone fragments were examined and the

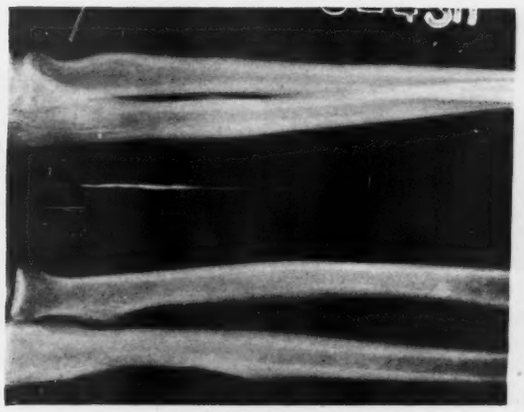

Fig. 2. Postoperative picture showing removal of nidus.

pathological diagnosis was osteoma. It is our opinion that the tissue examined was the perinidal sclerotic bone produced by the osteoid osteoma.

The patient made an uneventful recovery. A roentgenogram (Fig. 2) taken January 26, 1945, showed no evidence of recurrence. The patient feels well and has been relieved of his symptoms. He is back on active duty.

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Bookshelf Browsing

OLIVER WENDELL HOLMES

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OLIVER Wendell Holmes is immortal in American literature and renowned also in the annals of medical history. Few have attained his mark as a physician and poet. His life is one to be admired because of its contributions to the cause of humanity and to the esthetic enjoyments of peoples. His sincerity, honesty, and goodness could leave no man more secure in the hope for a peaceful rest under the eyes of God as the shadows of life grow longer.

Born in August of 1809 in Cambridge, Massachusetts, Oliver Wendell Holmes was the son of the Reverend Abiel Holmes, a Calvinist preacher.[1,21] His mother was Sarah Wendell, a descendent of Governor Simon Bradstreet and of the Puritan poetess, Anne Bradstreet.[6,9] A great believer in ancestry and proud of his descent from the colonial notables, Holmes was reared in an atmosphere of books and culture. It was at a very early age that he started to write poetry.

In 1824, at the age of fifteen, he attended Phillips Academy at Andover and remained there for one year.[1] In 1829, at the age of twenty, he was graduated from Harvard and received a bachelor of arts degree.[1,13,16] Evidently Holmes was undecided about his career at this point. He proceeded to study law for one year and then embarked on a medical career. He studied at Boston and Paris, and traveled through several European countries over a short span of time. In 1836, at the age of twenty-seven, he received the degree of doctor of medicine at Harvard; following this he joined the Massachusetts Medical Society.[1,13,16] Is it not interesting to note that six years before Holmes set out as a practitioner of medicine he had written a poem that has firmly implanted itself among the classics in American Literature—"Old Ironsides." The strength and vigor of its verses foreshadowed the power and perseverence which were to be contributed some thirteen years later to the fight for a great cause and a noble aspiration.

In 1838, Holmes was appointed Professor of Anatomy and Physiology at Dartmouth College and he held this post for two years. When he was thirty-one years old he married Amelia Lee Jackson, daughter of Charles Jackson, who was a judge of the Massachusetts Supreme Court for eleven years. They had two sons and one daughter.[1]

In 1836, Holmes wrote a paper called "Indigenous Intermittent Fever in New England." Six years later he published material he had presented as two lectures, "Homeopathy and Kindred Delusions," in which he classed homeopathy with the Royal Touch, The Weapon Ointment, and Sympathetic Powder.[14]

Shortly after Holmes was graduated from medical school he published his first volume of poems.[9] Successful as he was as a poet there seems to be a difference of opinion on the question of how successful a physician he proved to be. Perhaps the difference of opinion results from divergent interpretations of the meaning of success. Supposedly Bostonians had difficulty in reconciling his witty verse and conversation with the seriousness of his medical career. Nevertheless of his status as a poet, there is no question. He was the last survivor

of the "New England Group" of poets. During his career, Boston was the "Athens of America" and Holmes was wonderfully fitted to be its unofficial laureate.[9]

Later on in life, in the year 1874, Holmes wrote a letter to the New Orleans Medical and Surgical Journal in which he stated:

"I do not believe much in rhyming doctors, and since I have fallen into that way myself, I have had no professional homicide laid to my charge, and that for the best of reasons—I do not profess to practice medicine. In my opinion a man who has life and death, and the bodily and mental welfare of his fellow creatures in his hands, should have no ambition outside of his proper calling. To do his duty by his patients will tax all his powers. There have been a good many physicians who have also been poets and litterateurs, but very few instances that I can at once recall where they have really excelled in both ways. . . . Goldsmith and Smollett were great writers but medical failures."[2,3]

Mary Marshall quotes John T. Morse:

" . . . a brilliant career in the way of practice not only did not begin early with him (Holmes), but it never developed at all."[2,21] And Holmes himself said, "The great practitioners are generally those who concentrate all their powers on their business."

It must be stressed at this point that when Holmes claimed in the letter quoted above that he did not profess to practice medicine, he was sixty-five years old. Nevertheless fully thirty years before this he was engaged in his profession and at the same time had an established reputation as a rhyming doctor. Perhaps there have been only few instances in which men have excelled both as physicians and poets. Today the fact is unquestionably accepted that Holmes does fit into that category. One must differentiate between the great practitioner and the great physician. Holmes may not have been the former but he definitely is the latter. Supporting this view, Dr. William Osler, in a much quoted obituary address before a meeting of the Johns Hopkins Hospital Medical Society, proclaimed Holmes "the most successful combination the world has ever seen, of physician and man of letters."[18]

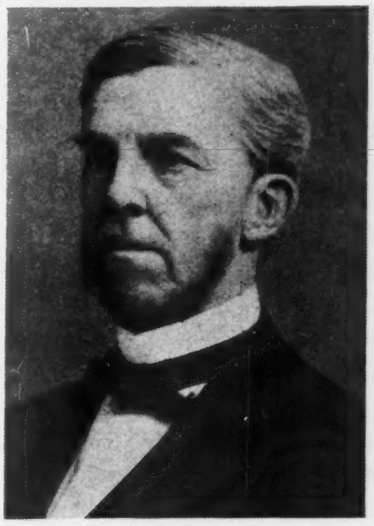

Fig. 1. Oliver Wendell Holmes.

In 1843, at the age of thirty-four, Holmes published his paper on "The Contagiousness of Puerperal Fever." This event was one of the most momentous in his medical career. It culminated a period of study and research, introduced a period of heated conflict and presaged an era of altered concepts in the treatment of cases of the dreaded puerperal fever. It is upon his work and teachings on this subject that the reputation of Holmes as a physician is based predominantly. Roy Finney remarks that this paper by Holmes is conceded to be the most important essay ever written by an American physician. "It is a product of that rare combination, a mind equally talented in both science and literature. Facts, arguments, deductions, arrayed as if by a master logician are expressed in the language of an expert in the art of rhetoric."[4]

Puerperal infection has probably occurred almost as long as children have been born.[8] The ancients regarded it as the result of the retention of the lochia and this explanation was accepted universally for centuries. References to puerperal fever are found in the writings of Hippocrates, Galen, and

Avicenna. In the early part of the seventeenth century, Plater claimed that it was a metritis essentially. In 1676, Willis wrote on "febris puerperarum" and the English term was probably used first by Strother in 1718. In the eighteenth century Puzos presented a milk metastasis theory. Many theories of the origin and nature of the infection were presented from the time of Plater until Semmelweiss proved the identity of the disease with wound infection, Pasteur had cultivated the streptococcus, and Lister had demonstrated the value of antiseptic technic.[8]

A prevalent theory in Europe for many years was that puerperal fever was due to miasmatic, telluric, or atmospheric influences. Even after Semmelweiss' book on the subject was published in 1861, this theory held its place. Hirsch studied the subject from a historical point of view and concluded that the disease was infectious, but the medical profession did not realize the importance of the disease as an infection until after the popularization of Lister's teachings and the recommendation of Stadfeld of Copenhagen that bichloride of mercury be used in obstetrical practice.[8]

Much credit belongs to Semmelweiss because his writings were based on personal investigations. Semmelweiss, however, was not the first to use a solution of chlorinated lime. Precedence goes to Thomas Alcock who used it in 1827.[8] In the United States the main indebtedness is to Holmes for introducing the idea that puerperal fever is infectious. Holmes' suggestions consisted of advising physicians to abstain from obstetrical practice for limited periods of time if they found that their patients were becoming infected. He suggested repeated bathing and changing of clothes between visits to obstetrical patients.[11]

The influence which Holmes' writings exerted were partially counteracted by the active opposition of Dr. Meigs and Dr. Hodge, outstanding figures in American medicine. The evidence collected by Holmes influenced them little and the observations on the contagiousness and epidemic character of puerperal fever made long before by Malouin at the Hotel Dieu in 1746, by Gordon at Aberdeen in 1795, and by Charles White of Manchester, England, in 1773, were of minimal significance as far as they were concerned.[8,10]

In his paper read before the Boston Society for Medical Improvement and published in the New England Quarterly Journal of Medicine, Holmes insisted that women in childbed should not be attended by doctors who had been doing postmortem examinations or attending to patients with puerperal fever. He said the disease could be transferred in this manner from one patient to another and that it could be transferred also to a patient from a case of erysipelas. A likely preventive measure would be washing hands in calcium chloride and changing clothes after attending a patient with puerperal fever.[11,15]

Twelve years after his first great medical publication the fight was still raging and there appeared another paper by Holmes which is listed among the medical classics, "Puerperal Fever as a Private Pestilence."[12] In this work Holmes repeated his views and referred to the work of Semmelweiss whose great achievement is evidenced by a mortality reduction among puerperal cases at the Allgemeines Krankenhaus in Vienna from 9.92 to 1.27 per cent. Yet at the same time Dewee's textbook, Diseases of Females stated, "In this country under no circumstance that puerperal fever has appeared hitherto, does it afford the slightest ground for the belief that it is contagious."[4,5]

In 1846, Morton demonstrated ether anesthesia on a patient on whom Dr. Warren of Boston operated successfully. The operation was for the excision of a vascular tumor. It was in a letter to Morton dated November 21, 1846, that Holmes suggested to Morton the terms "anesthesia" and "anesthetic" but he suggested consultation with an authority in the field before the terms were decided upon.[4,7,15] It is mentioned frequently that Holmes was the first to introduce this nomenclature. This

is denied by Sir William Osler, who in a talk before the Historical Section of the Royal Society of Medicine on "The First Printed Documents Relating to Modern Surgical Anaesthesia," said that the word "anaesthesia" is not a modern word but is encountered in Plato's "Timaeus" and was used by Dioscorides in the modern sense.

Holmes taught at the Tremont Street Medical School for several years and was connected with the Massachusetts General Hospital. In 1847, when he was thirty-eight years old, Holmes was appointed Parkman Professor of Anatomy and Physiology at Harvard. At the same time he was appointed Dean of the Medical School, retaining this post until 1853. It was years later, in 1871, that he relinquished the chair of physiology but he remained as Professor of Anatomy until 1882 at which time he resigned and was appointed Professor Emeritus.[1,13,14] Holmes was reputed to be an excellent lecturer, able, intensely interesting, pleasant, lively, and witty. Many people have been impressed by his metaphorical descriptions and Dr. Thomas Dwight said, "Medical readers will appreciate the aptness of likening the mesentery to the shirt ruffles of a preceding generation, which from a short line of attachment expanded into yards of complicated folds. He has compared the fibers connecting the two symmetrical halves of the brain to the band uniting the Siamese twins."[14] Holmes' attractiveness as a lecturer is apparent in his assignment to take the students in hand toward the end of the day when a less able speaker would have been unable to keep them awake.[13]

Of his attitude toward the nature of the work engaged in by the students no statement could be more enlightening than his own. He said, "We cannot wonder that the sensitive Rousseau could not endure the atmosphere of the room in which he had begun a course of anatomical study. But we know that the great painters, Michael Angelo, Leonardo, Raphael, must have witnessed many careful dissections, and what they endured for the sake of art, our students can endure for science and humanity."[18]

With the arrival of the year 1857 came the forty-eighth birthday of Dr. Holmes. As one writer has indicated, Holmes is noted as being one of the greatest of American authors, but had he written an autobiography at the age of forty-eight when the majority of men have made their place in the world he would have written mainly as a man of science.[16] During this year his papers called The Autocrat of the Breakfast Table were published in The Atlantic Monthly which Holmes was helping James Russell Lowell to edit. Holmes' success was immediate and the remainder of his career was settled. He was now an author rather than a physician and he continued to publish essays, novels, and poetry.[13] The Autocrat was followed by the Professor of the Breakfast Table and the Poet at The Breakfast Table. There appeared later Over The Tea Cups and three novels, Elsie Venner, The Guardian Angel, and A Mortal Antipathy. It was the Autocrat which established Holmes permanently in American literature. Succeeding years saw additional literary endeavors and successes. Holmes published a biography of Motley, the historian and diplomat, and also a life of Emerson. After touring Europe in 1886 he wrote One Hundred Days in Europe and this was received well. Verses appeared rapidly, and each year for thirty-eight years from 1851 to 1889 Holmes wrote a poem for the reunion of his class at Harvard. Of his best known and still popular poems are The Last Leaf, The Chambered Nautilus, and The Wonderful One-Hoss Shay.[16] It was William Osler who suggested as a bed-side library for medical students ten works which he deemed worthy and sufficiently great to occupy the shelf. Tenth on that list is Oliver Wendell Holmes' Breakfast Table Series and it takes its place alongside the Old and New Testament, Shakespeare, Montaigne, Plutarch's Lives, Marcus Aurelius, Epictetus, Religio Medici, Don Quixote, and Emerson.[17]

In 1863, Holmes was University lecturer for one year and from 1876 to 1882 he was Overseer of Harvard College. Between 1880 and 1889 he received many honorary degrees: An LL.D. from Harvard and from Edinburgh, and Litt.D. from Cambridge, a D.C.L. from Oxford and an A.M. again from Harvard.[1,13]

When Holmes was eighty years old he was asked by William Osler whether he would rather be known to posterity as the man who in this country aroused the medical profession to the dangers of puerperal sepsis as an infectious disease or whether he would prefer to be remembered as the author of The Chambered Nautilus. Holmes' reply was, "There is some selfish pleasure to be had out of the poem, perhaps a nobler satisfaction from the life-saving labour . . . "[19] Probably recalling at this time his grand fray with the opposition of Meigs and Hodge, a statement he once made might very well be appropos: "To reach a port, we must sail, sometimes with the wind, sometimes against it, but we must sail and not drift, nor lie at anchor."[20]

In 1894, at the age of eighty-five, Dr. Holmes died painlessly while sitting in his chair. His life was full in years, in work, in achievement, in satisfaction, in goodness, and in fame. As applies to his spirit, no words could be better applied than his own: "To be seventy years young is sometimes far more cheerful and hopeful than to be forty years old."[18]

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SURGICAL HOSPITAL*

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FOR ten days to two weeks we are in the doldrums, bivouaced in a corktree grove, sitting around writing letters, washing clothes, sketching, studying anatomy and just rumor-mongering or speculating on our next move. During this time there has been increasing heavy gunfire in the distance and frequent commutation of plane squadrons overhead to and from the front. We are in the hills and at night the sky over the mountains flashes intermittently with brightness that is not lightning.

Then comes the alert we have all hoped for and yet dreaded. We are up at 5:30 A.M. for a messline breakfast of powdered eggs, hardtack and coffee. After that valpacks are strapped, air let out of mattresses, beds rolled and strapped tight with much heaving and sweating, tents struck and all loaded with convoy ready to move sharp on the dot of nine as ordered. The usual happens, however, and we sit in the loaded trucks till noon before starting. Perhaps you do not have a clear picture of what a surgical hospital, when on wheels, looks like. It takes over 100 two-and-a-half ton trucks, ambulances, weapons carriers, carryalls, command cars and jeeps to transport just this one outfit of some fifty officers, sixty nurses and three hundred enlisted men with equipment. Of course all those vehicles do not belong to us; some of them do but the rest have to be assembled from other outfits to move us. Maintaining the required interval of 200 yards between vehicles on the road, our convoy alone covers over ten miles.

Officers are called together, told the route and each assigned to the cab of a two-and-a-half to ensure keeping intervals and to avoid getting lost should we happen to mix up with other convoys on the road. I ride the truck carrying operating room equipment and have a pleasant colored boy from Georgia as my driver. The inside of his cab is studded in typical soldier fashion with pictures of beautiful girls in the nude, semi-nude or simply Hollywood sweaters. He is good fun, always humming or singing, and during stops he runs ahead to get cans of grapefruit juice and boxes of graham crackers from the ration truck which, as luck would have it, immediately precedes ours.

Lunch apparently has not been planned but I had been foresighted enough to pick up some cans of C-rations so we make out with cold stew, hash and "iced" coffee. The latter is produced by dissolving the ration powdered coffee and lump sugar in canteen water, mixed with lots of imagination. Rather lumpy but good nonetheless.

The trip is slow, dusty and hot. Cold as it is in the mountains of North Africa, the sun gives you a good going over from nine to four. We form only a small link in an endless chain that bumps along in white clouds of choking dust, stops and waits every little while and then bumps along again with much shifting of gears and swearing of driver. One pause lasts over an hour in shimmering heat waves while a whole convoy of heavy guns rolls slowly past round the curve and up the hill, the gun crews white with dust clinging precariously to their perches on trucks and carriages. At each stop flies swarm over us, vicious insects with bites that sting. Another pause and a division of engineers goes by, queer looking trailer trucks loaded with heavy bridge sections all ready to be slapped together when needed. As each vehicle passes we automatically hold our breaths and then fill our lungs with only half dusty air before the next comes along.

*This has been excerpted from a diary that was kept throughout the war by the author.

This is all country which has been bitterly fought over only a short time before. The road is rough and pocked with bomb holes which have hastily been filled in. All along on either side are huge and small craters and here and there a lorry or other vehicle which has been burned or smashed and pushed off the road. Some are wrecks which had simply gone off a turn and over the bank in the blackout. It is evident that both Americans and British have been through here. Signs such as "Brooklyn Bridge" and "Pall Mall" have been stuck up on crude posts or scrawled on the walls of shattered buildings.

At one place we pass a dozen Mark VI tanks which have been shot up. Some of the tanks are completely overturned, some up-ended, some on their sides, some with three-inch shell holes through four-inch steel. Most of them are burned out. Incidentally, it was in one of them that I had been strafed by three Messerschmitts only a few days previously. I had gone up to look through them for parts I needed in building a suction pump for the operating room. I had just climbed into the least damaged tank and was having a wonderful time elevating and depressing the cannon and wheeling the turret around from the control tower when I heard the planes coming. They did not sound like ours and furthermore the ack-ack set up a terrific clatter so I dove for the bottom of the tank without looking up, figuring it was the safest place I could find. I just made it as my two enlisted men piled in on top of me. Then came the crump-crump of bombs and chatter of machine guns but fortunately nothing landed nearby. It was only afterwards I noticed the shelves around the inside of that tank were loaded with unused cannon shells.

About 5:30 P.M. we reach our destination, a beautiful site on a gently sloping hilltop in the open, midway of a long valley ringed on all sides by mountains. The latter are arid, rockridged and treeless. The wheat fields through which we bump and jog off the main road have obviously been used recently by tanks, artillery and infantry. Tracks run off aimlessly here and there and foxholes are numerous, each supplied with its telltale mound of empty ration tins, ammunition casings, waste litter and blackened stones. Down the hillside from us lie the tragic remains of an allied bomber where it had crashed and burned, engines, propellors and bits of wings scattered in all directions.

The first part of the convoy has already set up the O.R. and the mess tent when we arrive. The generator is hooked up and going. The place seems a bedlam of grinding trucks and shouting confused men. But the general layout of areas for operating rooms, wards, living quarters for officers, nurses and enlisted men has been planned and in remarkably short order a tent city springs up as though by itself with unseen power. The C.O. says we are due to start receiving cases at six. I think to myself, "Yes, yes, I know—that means we probably won't see a thing till tomorrow." So I slip off to my pile of baggage and get messkit and canteen cup. Chow is already being passed out but right on the dot of six and before I can eat a sergeant comes running to say that we have received a half dozen wounded, two major cases among them, and other ambulances are arriving. This is certainly one time the army knows what it is doing and does it.

So I check on shock treatment for the two worst cases, a belly and a shattered knee. Both have had plasma forward and need blood. While the transfusions are running I get my stew and coffee. Then to work: a chest wound to be taken care of, a belly explored, fractures for reduction and splinting after *x*-ray, jagged wounds to be cleaned up and hemorrhage controlled and burns to be dressed under anesthesia. Now and then an extremity is damaged beyond hope of saving; somehow these seem the most pitiful cases. Patient after patient is brought in; six tables are filled all the time; an endless procession of new faces and injuries. "What's the matter with you, lad? Hurt badly?" "No, Sir,

I'm all right; just a bit of shrapnel from an 88." "You'll be O.K. in a jiffy—here, let's have a look at it; we'll put you to sleep first."

A pause for hot coffee and food at midnight; then we go back at it. We lose all track of time. The long tunnel of operating tent flooded with light, the smaller tent leading to it where we scrub and patients silently wait their turn, stretchers side by side on the canvas covered dirt floor, the trickle of cold water over our bare arms and the sting of bristles on raw skin, the sea of white drapes beneath caps and masks bent over tables, the hasty cutting away of clothing caked with dirt and clot, the stickiness of blood partially dried on fresh wounds, the sight of marble skin surrounding islands of scarlet or innocent looking puncture wounds revealed as the filth is removed—all make a confused picture that seems unreal; and yet here we are, here I am, a part of it, going on and on.

We are working under blackout; and such pitchblack darkness it is. It has commenced to rain and in a short time the ground is a slippery, slimy sea of clay which clings in great sticky knobs to shoes. Flashlights are banned; we are near the front now. Poor litter bearers struggle through the dark with their loads from receiving to *x*-ray to O.R. More loads, often heavier due to plaster casts, are carried from O.R. to wards. Stumbling and slipping, shoulders strained, muscles long since numb with fatigue, these men carry on. Tents are invisible in the black; one works by instinct in a seeming void, chilled and wet to the skin. "Hey, is this *x*-ray?" "No, dope, its Surgery Three." "Well, where the h . . . is x-ray then?" "Dunno, Joe, try second tent in the next row." And off again they go with heavy burden.

Long lines of ambulances loaded with torn humanity, their dim lights like pale watery eyes peering through the rain, inch forward bumper to bumper to discharge their pain-ridden cargoes into overflowing receiving tents and be off again. Quick, deft examination of the endless stream of wounded follows; these are sent to shock ward, those to O.R. stat, others to *x*-ray; there is no time to ponder; the lightly wounded go directly to wards for hot coffee, soup and food. "Man, my first meal in three days and is it good!"

In another tent a group of surgeons work silently and swifty to take blood for countless transfusions. A pool of volunteer donors from supply services is already on hand; as soon as some have given others take their places. Blood flows through the laboratory to the shock ward and O.R. in a steady life-giving stream.

I am off duty and due to get some rest the next evening when the C.O. greets me at supper: "Come and see a case with me; tell me what you think ought to be done." A lad of twenty, toxic and delirious, strafed by a plane two days before, has a compound fracture of both ankles. His feet are swollen and discolored, the wounds green and stinking, with telltale bubbles beneath the skin—gas gangrene. He needs attention and in a hurry. Transfusions are started, anti-gas gangrene serum and sulfonamides given intravenously in massive dosage, along with fluids and glucose. Blankets and hot water bags are used for shock. Meanwhile a separate pyramidal tent is set up for isolation; a stretcher serves for a table and within an hour both legs are removed below the knee. A special nurse and ward boys are assigned but the patient is too sick for me to leave; he is still toxic, delirious and violent as reaction comes from the anesthesia.

The needle clogs; the veins are thrombosed and the low pressure, due to shock, keeps them collapsed. I try to get into another vein but cannot; I cut down and expose another but that is clotted solid. The third try succeeds but it is all we can do now to hold his arm steady and keep the needle in the vein. It is early morning and the patient is restless and screaming. Morphine does not touch him, nor does sodium amytal. He does not seem so good; he is paler and colder. A glance beneath the

blankets reveals a secondary hemorrhage; the dressing on the left stump is soaked with fresh blood. An emergency tourniquet is quickly tightened; fresh instruments and dressings are brought from the O.R.; the stump is bared, the bleeder found and sutured and a new dressing applied.

Over and over I keep thinking, "Why bother—the kid can't make it anyway—he's cooked—no blood pressure, pulse like a trip hammer and barely felt at that—respirations way up—skin like parchment—nose pinched and nostrils sucked in with effort to breathe—lips blue—hands cold as ice. Besides, if he does he'll be a cripple for life." I cannot shake these thoughts but I keep on, automatically, as one possessed. Oxygen is tried but he does not get much; he is too violent and will not tolerate the mask. Another transfusion is given while the needle remains in the vein.

It is bitter cold in that tent and there is no means of getting heat. An overcoat helps but it gets in the way. Heavy thunder storms and pouring rain continue unabated outside; it makes an incessant rattle on the canvas. There is a call for me—I am wanted in the O.R. This time they will have to do without me; someone else will have to take it for I am not leaving. A major comes over; there is a bad case, compound fracture of both thighs, to be done. "OK, get so-and-so; I'd like to help you but I can't drop this yet."

Three days later our lad is still with us and for the first time he asks for water. What a wonderful sound those few words made; I'll never forget. He has gone further back now and I do not know what has happened to him but I'm sure he made the grade. What a tragedy the loss of his feet but he will walk again and, as we told him, it is not as bad as an arm.

Then comes a glorious spring day, with bright sun, cloudless skies, wheat fields ruffled by a grateful breeze and a yellow green countryside deceptively peaceful. Suddenly there is a loud explosion and heavy smoke rises in an inverted cone from a field nearby. Arabs, horses, cattle and sheep scatter in all directions, their paths clearly marked by fresh furrows in the waist-high grain. A land mine has been touched off. Soon details are known: Three Arabs have been killed outright and there are five others for us to patch up. There is nothing unusual about this. The fields were sown with mines by the Germans. Now that the Arabs are returning to their homes accidents are bound to happen. Unused hand grenades, left behind in the rush of advance, are devil's bait for Arab kids—children with knees blown through, hands mutilated, faces crushed—a bloody toll of the innocent.

One week passes and the heavy rumbling that has formed a part of our life day and night since arrival has dwindled in the distance. Admissions taper off. In two days no more casualties appear. Bizerte is taken, Tunis has fallen, the campaign in North Africa is at an end and we are ready to move.

EVOLUTION OF SURGERY

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SURGERY has rightly been called the Queen of the medical arts. Its history is also the history of man himself. Even the most primitive races had a surgical lore of sorts. The first surgery of mankind was probably that of extracting arrows received in combat and healing the wounds incidental to hunting. As the infliction of the elements and less friendly living creatures grew more relentless a more complex surgery was evolved. The skull in cases of severe headache was opened; an impressive collection of Peruvian skulls, plainly bearing evidences of trephening, speak eloquent testimony to primitive surgical skill. Surgical instruments, fashioned of stone, have been unearthed, and modern surgeons have demonstrated by actual experiment, the feasibility of working with such instruments. Moreover, paleolithic art has preserved the earliest evidences of surgical craft in the caves of Europe, where drawings representing hands that have been mutilated either accidentally or surgically still exist. Some of these drawings represent hands from which one or more of the fingers have been removed, suggesting injuries received in the handling of the rude implements then in use or amputations as the result of such injuries.

The remarkable researches of Dr. Marc Ruffer in paleopathology which he carried on upon ancient Egyptian mummies reveal an astounding knowledge of surgery among the ancient Egyptians. Many of the surgical procedures heralded in later centuries as new were long ago known to the Egyptians. Eye surgery, especially, attained a high plane of development. The abdomen was opened in diseased conditions and the abnormality skillfully corrected. They, moreover, had great skill in opening the skull and this method of therapy was used in curing migraine and epilepsy. They also performed circumcision and removed superficial tumors.

Greece, in somewhat later times, became fertile soil in which the art of surgery grew lustily and flourished. The fifty years preceding the birth of Hippocrates, the Father of Medicine, was a favorable one. There had come a sudden rush of expansion among the Greeks to the west. The Persians in the east were extending their boundaries and rolling forward to the Mediterranean those vast armies which the Greeks crushed at Plataea, Salamis and Mycale. During this time military surgery expanded marvelously and enriched the experiences of surgeons who used their newly acquired knowledge and skill for the benefit of the citizens as well as for the soldiers.

Close upon the heels of this important surgical era came that of Hippocrates whose achievements in surgery no less than in medicine helped to establish the early science on a firmer footing. This Goan treated with success diseases of the bones and joints, ulcers, fistulas and hemorrhoids. He trephined the skull, reduced hernias and repaired defects that Nature had left in too hurriedly completing a job. He removed stones from the bladder with signal success, and he was among the first to describe artificial collapse of the lungs and to open and drain the chest in cases of inflammation and pus formation in the lung coverings.

After Hippocrates there was no notable name in surgery for five hundred years. Galen, of Alexandria, made his appearance after this lapse of time, and his contributions to the healing art gave it new prestige. Perhaps his greatest contribution to surgery was the ligaturing of arteries. Moreover, he devised a new treatment for weakened artery walls. He was especially interested in the nervous system upon which he carried out many experiments through

which he originated many new surgical procedures for the cure and relief of injuries to the brain and the spinal cord.

The Middle Ages furnished a wonderful array of names: Bruno da Longoburgo, Hugh, of Lucca, William, of Salicet and Lanfranco. Those of western Europe were Henri de Mondeville, Guy de Chauliac, John of Ardern, and John Yiperman. Bruno helped to clarify the meaning of the term surgery; he wrote a code of ethics for surgeons to observe. In 1252, he published his great work "Chirurgia Magna" in which he advocated several surgical innovations. He insisted that the essentials of surgery were to bring separated parts together, to separate those that have become abnormally united, and to extirpate what is superfluous. Bruno advocated the dry treatment of wounds and devised several interesting procedures in abdominal surgery. If there was difficulty in bringing about the reposition of the intestines, he advised that they were first to be pressed back with a sponge soaked in warm wine. If the omentum, the apron-like structure in the abdomen, happened to squeeze its way out of the wound, all that was black and green, must be removed. In cases in which the intestines were wounded they were to be sewed with a small needle and silk thread, and care was to be exercised in bringing about a complete closure of the wound.

Following Bruno the next surgeon of importance was Hugh of Lucca. His most important contribution to surgery was anesthesia. Mandragora served as the base for an anesthetic in those days, though a combination with opium was the favorite. Da Lucca's method was by inhalation, and this means of administration was used for fully a century; it consisted of saturating a natural sponge with a solution of madragora, opium, hyoscymamus, lettuce, camphor and nenuphar. This anodyne was dried and when the occasion for using it arrived it was moistened with hot water and held to the patient's nostrils until he fell asleep. Lucca was among the first to observe strict cleanliness in the treatment of wounds. He avoided the use of the probe and employed compresses soaked in wine.

By far the greatest surgeon of the thirteenth century was William of Saliceto. He, like Lucca, had considerable experience in military surgery, and the outcome of his wide experience and researches was a systematic work on surgery. He set down in detail the treatment of various sorts of wounds, the suturing of nerves and intestines and the treatment of fractures and dislocations. He distinguished between hemorrhages from the arteries and veins. His method of treating water on the brain is of particular interest. He rejected opening the skull and recommended instead puncturing the scalp and membrane by a cautery, a very small opening being made and the fluid being allowed to escape only drop by drop. Saliceto's discovery tended to popularize the use of the cautery in surgery.

As Saliceto was the most distinguished surgeon of the Thirteenth Century Lanfranco was the foremost surgeon of the Fourteenth. He was exiled from Italy and came to France to live where he produced his "Chirurgia Magna." He argued that it was silly to fix a line between the work of the surgeon and that of the physician and between practice and theory. He gave a good account of the symptoms of fracture of the skull, and was the first to describe concussion of the brain. He recommended ligature as a means of stopping hemorrhage, and maintained that exposure to the air favored the formation of pus in wounds. He advised cutting the nerves in cases of traumatic tetanus. A thing for which he was noted was his method of stitching nerves completely divided in cross-section. This he said was the surest method of reintegrating the nerve. Not only was the nerve itself rehabilitated by this method, but after the operation the restoration of usefulness to the member involved was more complete.

The foremost pupil of Lanfranco was Henri de Mondeville who, above all,

pointed out the necessity of a friendly relationship between physicians and surgeons. Unlike his master he differentiated between theory and practice, and he insisted on a practical acquisition of surgical knowledge rather than theoretical. He advocated moderate boldness and did not countenance radical surgical treatment. He was impatient with the surgeon who was ever ready to employ surgical means when medicines would do as well. He evidently was not very satisfied with the state of the surgical art of his day, for he remarked: "Many more surgeons know how to cause suppuration than to heal a wound."

The most brilliant contemporary of Mondeville was Guy de Chauliac, who, in many respects, was as great a surgeon as Lanfranco. He, like Mondeville, insisted on experience rather than on authority. In cases of fracture of the leg, in addition to splints reaching to the foot, he employed a box or truss of straw to support the limb and attached to the foot a lead weight by means of a cord passing over a little pulley. He advocated that cancer be treated at an early stage and preferably with the knife. He made use of the speculum in certain obstetrical cases, and he gave an account of a ceserean operation. His special interest lay in the radical cure of hernia, and he devised six different operations for that purpose.He, moreover, developed a method of taxis by which to reduce hernias. He adopted stiffened bandages of various sorts, especially employing white of egg for this, and sometimes moulding bandages to the limbs in cases of fractures.

John, of Ardern, and John Yperman were among the later medieval surgeons of note. Ardern described rabies and drew an accurate picture of that disease. He also knew the clinical manifestations of traumatic epilepsy and suggested treatment. Yperman was well acquainted with and treated cases of dropsy, rheumatism, apoplexy, epilepsy, lung abscess, hemorrhage, liver abscess, blood-spitting, hardening of the spleen, and a host of other surgical diseases.

The greatest surgeon at the dawn of the Renaissance was Ambrose Paré. "Mere knowledge without experience," he said, "does not give the surgeon much self-confidence." All his own knowledge was acquired through experience, first as a dresser at the Hotel Dieu and then as a military surgeon. He treated with success wounds inflicted by sword, lance, halberd, stone arquebus, pistol, culverin and other types of firearms. He described various forms of fractures, including fractures of the leg, the arm and the skull. He was the inventor of arterial forceps, many different kinds of surgical instruments as well as artificial limbs and artificial eyes. Paré suggested that syphilis was the cause of weaking of the walls of the great arteries of the body. He was the first to use wire sutures to treat harelip. He removed concretions from the joints, improved the method of trephening, and made remarkable advances in the surgery of the eye. However, he is most known for his improvement in the treatment of wounds and because he introduced the ligature in amputations. In former times custom prescribed that wounds should be treated with boiling oil. Paré hit upon the idea, quite accidentally, of utilizing an extemporized simple ointment instead of boiling oil. His men recovered much more rapidly under this system. His introduction of ligaturing arteries instead of using the cautery abolished the ancient dread of bleeding to death and saved many lives.

Doctor Saleeby has said that the history of surgery may well be divided into two periods: Pre-Listerian and Listerian. During the interval that elapsed between Ambrose Paré and Lister surgery was enriched by the contributions of a remarkable group of men: Borelli, the physiologist, Malphighi, the anatomist, Paracelsus, the chemist, Van Helmont, who elaborated the teachings of Paracelsus, and Harvey, who discovered the circulation of the blood The pre-Listerian period was characterized by the presence of pyemia, septicemia,

erysipelas, and hospital gangrene which were certain to follow surgical intervention. With the introduction of ether and chloroform, operations increased in number and range. The mortality caused by the above mentioned infections was appalling. Lister particularly concerned himself with the problem of controlling these diseases, and with his appointment as professor of surgery at the University of Glasgow he buckled down for a practical solution of this problem. His special interest lay in the study of suppuration in relation to decomposition. From previous experience with carbolic acid as a deodorant and destructive agent in entozoa, it occurred to him that carbolic acid might prevent suppuration in cases of compound fracture. The first attempt to use carbolic acid in the treatment of compound fracture was not successful; in August of the very same year, however, this new method was employed with complete success in a case of compound fracture of the arm. Other successes followed this one and within a period of two years Lister's wards in the Glasgow Royal Infirmary became the healthiest in the world. At first he had prejudice and ignorance to fight, but in time the greatest surgeons in Europe were won over to antiseptic surgery. First came Thiersch and Richard Volkmann, of Germany, then Billroth, of Vienna, and Lucas-Championniere, of France. With these leading surgeons accepting the new doctrine the practice became widespread, and a wonderful new era in surgery was ushered in. Operations which no one would have dared to have undertaken because of the almost certain fatal outcome were now attempted with impunity.

Among the most notable operations in the dawn of Listerian surgery is that of Sir Victor Horsely for the excision of the Gasserian ganglion in neuralgia. This operation has been of great value in restoring elderly people to a life of usefulness, whose existence previously had been one of pain and misery. This operation could not have been undertaken in pre-Listerian days without certain death; yet today the skull may be opened, a large area of the brain exposed and turned aside and the Gasserian ganglion removed. Today 10 per cent of brain tumors are amenable to surgical treatment with good prospects of full recovery in a majority of cases.

The present age is so important in surgical history that the student of medical history in the future will have more to say about the present era than students of today have to say about all preceding periods. The World War served as a powerful impetus. Surgery was put to its severest tests and had to advance at a lively pace. Blood transfusion as a therapeutic measure for the combatting of shock and hemorrhage was widely resorted to. Methods of anesthesia were improved and facilitated. So was radiography and other means of determining before operation the nature of the injury and the exact location of bullets, shrapnel, and other foreign bodies. Small fluorescent screens that could be closely applied to the skin of the patient were used. Fractures and other injuries that could not be detected on the screen were often revealed by an examination of the plates. In not a few cases sterescopic radiographs proved of great value.

Some of the most notable results were achieved in plastic and kineoplastic surgery. The restoration of faces of soldiers mangled by instruments of the most fiendish design of modern warfare evoked the most popular admiration. Quite a few years back it was something of a feat to restore a nose lost by disease or accident. Now it is an everyday occurrence. Various parts of the body are now restored with a wonderful degree of nicety. Plastic surgery has made progress in leaps and bounds.

Commenting on the progress that Listers discovery has made possible in surgery, the late Dr. William Williams Keen made note of the following: "Amputations have decreased. Instances which in pre-Listerian days would invariably demand amputation are now quite possible of recovery without amputation. External tumors are now

removed without fear of erysipelas from all parts of the body. Even in cases where the tumor is internal there is no hesitancy about removing it. Opening the skull for curative and exploratory measures is now more frequent. Abdominal operations which would have been formidable several decades ago are now commonplace. Today the brain surgeon attacks tumors of even so inaccessible a place like the pituitary gland at the base of the brain, punctures the lateral and fourth ventricles of the brain, successfully extracts foreign bodies and in some cases relieves epilepsy and mental disorders. In the neck, goitres of any size are removed; in the chest the heart itself is sutured for gunshot and stab wounds, treated surgically for severe types of heart disease; the esophagus is treated for cancer; large portions of the chest wall are removed for treating cancer of the lungs."

The abdomen may be opened and various operations performed, even the total removal of the stomach, without the least danger. The intestines may be opened, anastomosed and short-circuited. Tumors of the liver, gallstones and gallbladder may be removed with ease. In the pelvis the bladder may be opened, rebuilt when it is defective and even wholly removed when it is hopelessly diseased.

Very impressive advances are being made in transplantation surgery. Skin, bones, joints and even half joints are transplanted with success. The trend in this direction is to transplant organs from healthy individuals to those whose organs have been destroyed by disease. Remarkable instances are the transplantation of testicles and thyroids for rejuvenation. Nerves are sutured and anastomosed. The same is true for blood vessels. The blood stream is criss-crossed to prevent gangrene.

Surgery today is veritably a super-surgery. There is no problem too difficult for the modern surgeon to tackle. The accomplishments of recent surgery thread a story full of wonder.

DOCTOR WATSON, I PRESUME?

R. P. GRAHAM
Assistant Professor of Chemistry, McMaster University
HAMILTON, ONTARIO

WATSON designates himself in the explanatory sub-title of "A Study in Scarlet" as John H. Watson, M.D. This is his signature to the Preface of "His Last Bow," and it is part of the inscription painted on the "travel-worn and battered dispatch-box." His first published work opens with the words: "In the year 1878 I took my degree of Doctor of Medicine of the University of London." By virtue of such evidence no one appears to doubt that Watson was the holder of a doctor's degree in medicine. But the matter is open to the most serious question.

In "The Field Bazaar" Sherlock Holmes deduced that a letter Watson had received from Edinburgh University was an unofficial communication: "This I gathered from the use of the word 'Doctor' upon the address, to which, as a Bachelor of Medicine, you have no legal claim." That would seem to settle the matter of the doctorate. No doubt there will be objection raised to this on the ground that "The Field Bazaar" is not canonical. But in text that undeniably is Watsonian, "The Adventure of the Dying Detective," Holmes bluntly tells Watson that he has only "mediocre qualifications" as a medical man. Could the M.D. from London be described as "mediocre"? Certainly not; this degree is an extremely difficult one to obtain.[1]

In further proof, if it be needed, of the spuriousness of Watson's alleged degree, it may be mentioned that the University of London has no record of a John H. Watson taking the M.D. in 1878. And if it is unlikely that Professor Moriarity caused the record of Holmes at Cambridge to be obliterated,[2] it is still more improbable that he would trouble himself about Watson's academic history.

Even if Watson's medical degree was not what it might have been, he earned, many times over, a doctorate of letters. No one begrudges him the use of a title, but he should not have tried to deceive us. "What is the meaning of it, Watson?"

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2. SAYERS, DOROTHY L. Holmes' College Career. P. 28. In Baker Street Studies (edited by H. W. Bell). London, 1934. Constable and Co.

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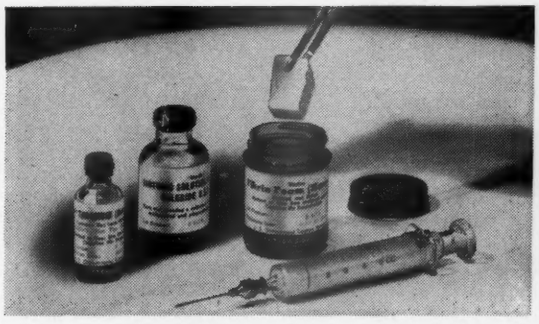

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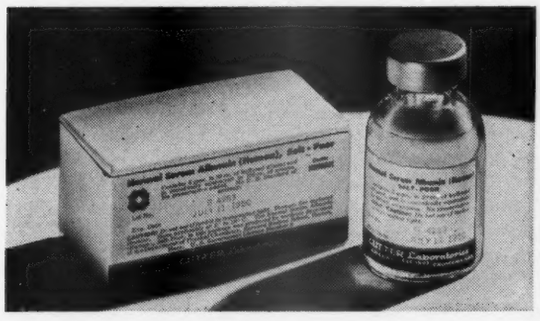

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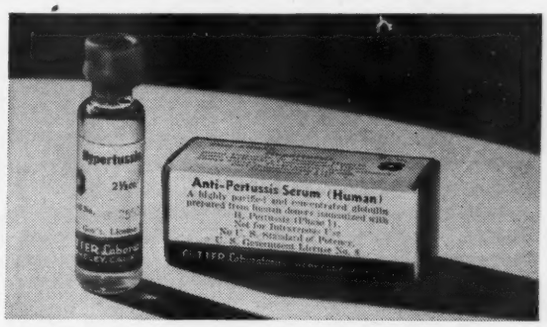

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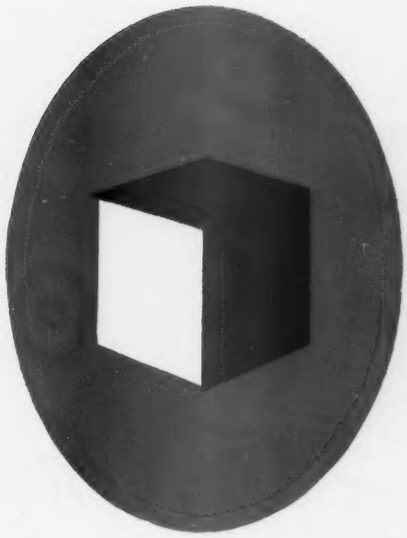

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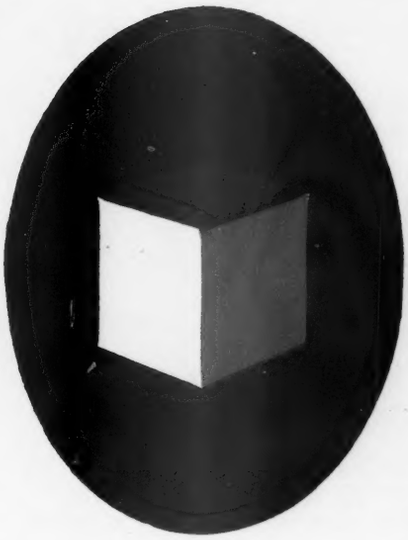

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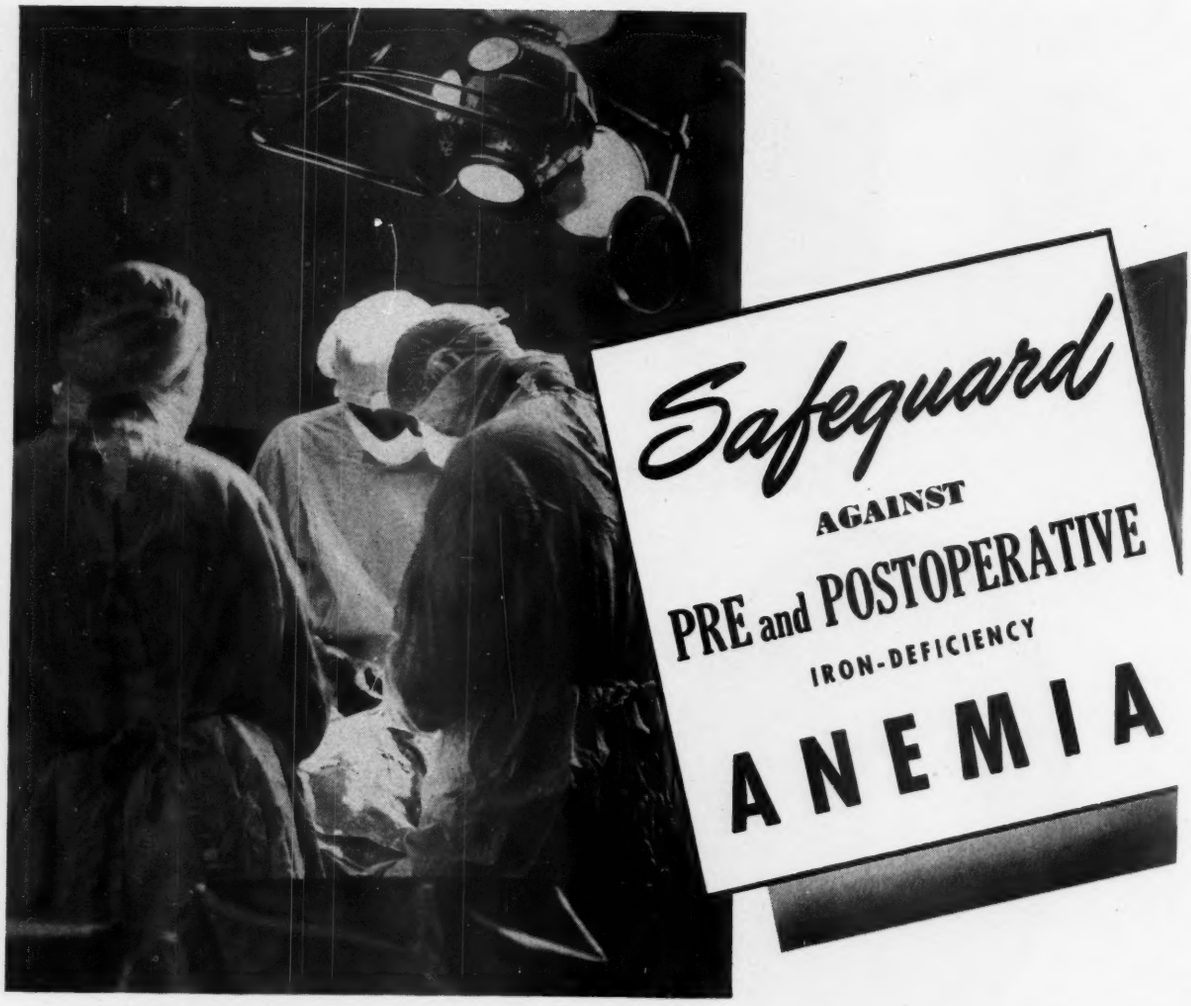

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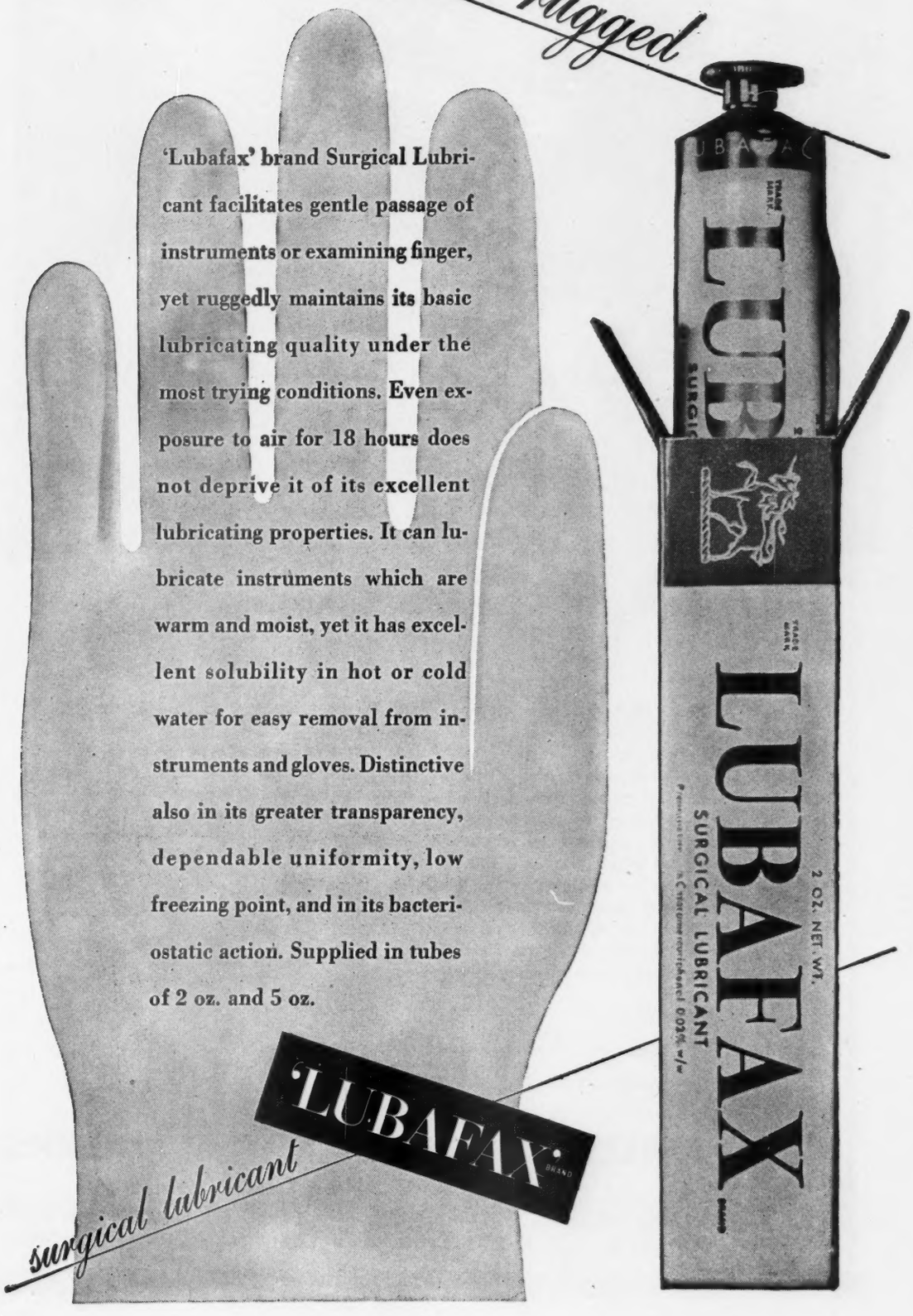

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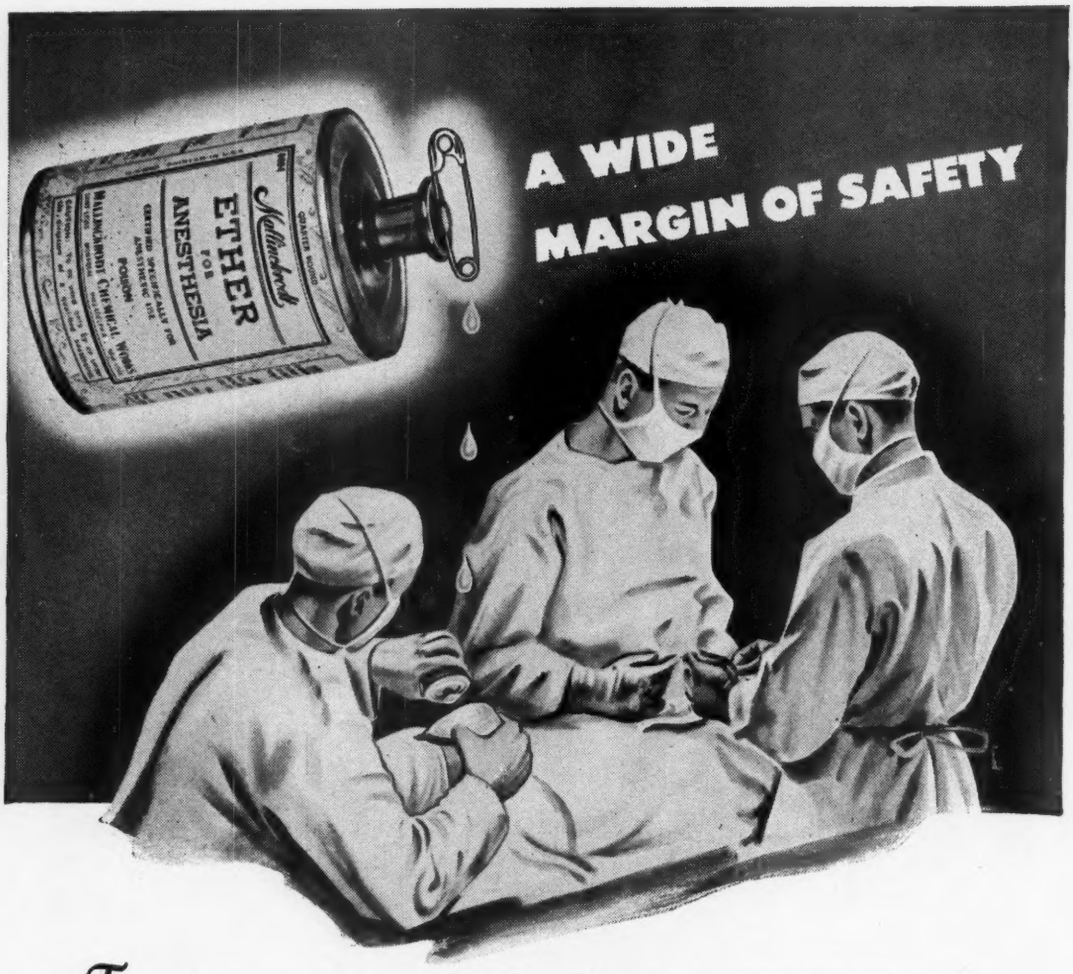

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1 Editorial: Anesthetics Old and New, Anesthesia and Analgesia, 23:44, Jan.-Feb. '44
2 R Blair Gould: Anaesthesia for the Patient in Shock, Anesthesiology 5:129, March, '44.
3. Major C F McCuskey: Anesthesia At The Front, The Military Surgeon, April, '44

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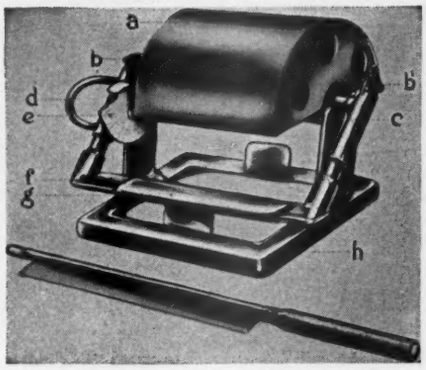

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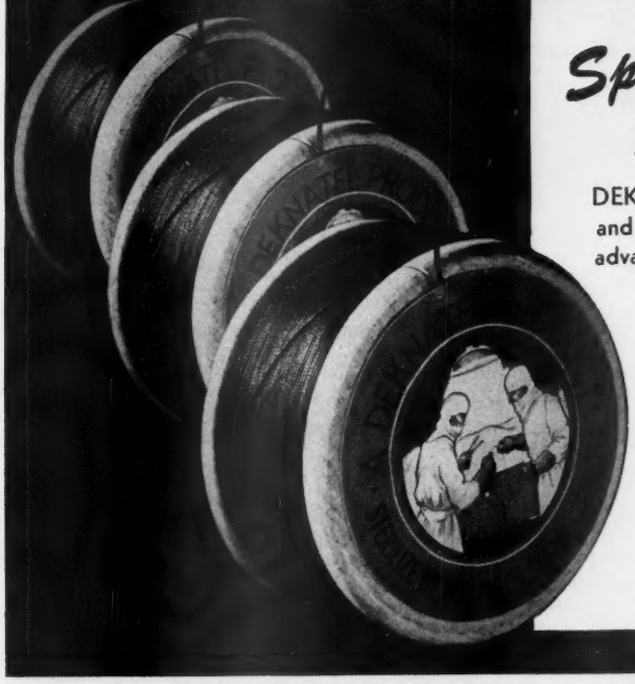

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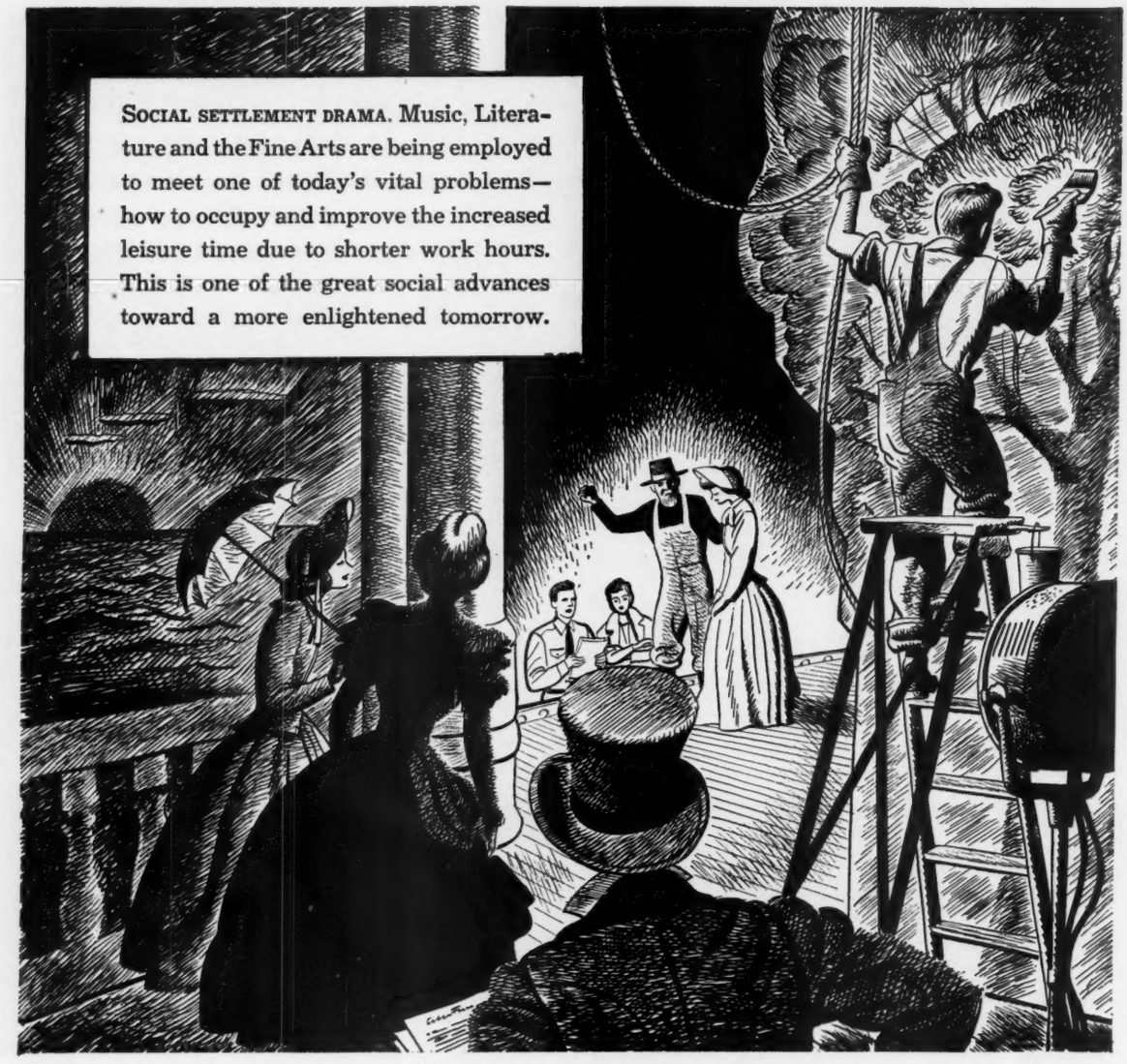
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After the skin has been closed and the patient returned to bed, healing of the operative wound and ultimate recovery depend upon a multiplicity of factors. Foremost among these is the nutritional state. Ample evidence makes it clear that during the period of postoperative surgical recovery, the metabolic need for protein and vitamins is sharply increased over normal requirements. Failure to satisfy this demand results in retardation of the many processes involved in recovery.

For these reasons, postoperative feeding is begun, and resumption of normal eating habits is encouraged, as quickly as possible. Hence breakfast becomes an important meal to the postoperative patient, not only to satisfy hunger but primarily because of the contribution it can make to total daily intake of essential nutrients.

Cereals enjoy an important place in the postoperative breakfast. Because of the large variety available, taste appeal is assured. Whether eaten hot or cold, together with milk and sugar, the cereal dish provides biologically adequate protein, significant amounts of essential vitamins, minerals, and caloric food energy. The table of composite averages below indicates the nutritional composition of the cereal dish composed of 1 ounce of cereal (whole grain, enriched, or restored to whole-grain values of thiamine, niacin, and iron), 4 ounces of milk, and 1 teaspoonful of sugar.

Calories	**202**
Protein	**7.1 Gm.**
Fat	**5.0 Gm.**
Carbohydrate	**33 Gm.**
Calcium	**156 mg.**
Phosphorus	**206 mg.**
Iron	**1.6 mg.**
Thiamine	**0.17 mg.**
Riboflavin	**0.24 mg.**
Niacin	**1.4 mg.**

The presence of this seal indicates that all nutritional statements in this advertisement have been found acceptable by the Council on Foods and Nutrition of the American Medical Association

CEREAL INSTITUTE, INC.
135 SOUTH LA SALLE STREET · CHICAGO 3

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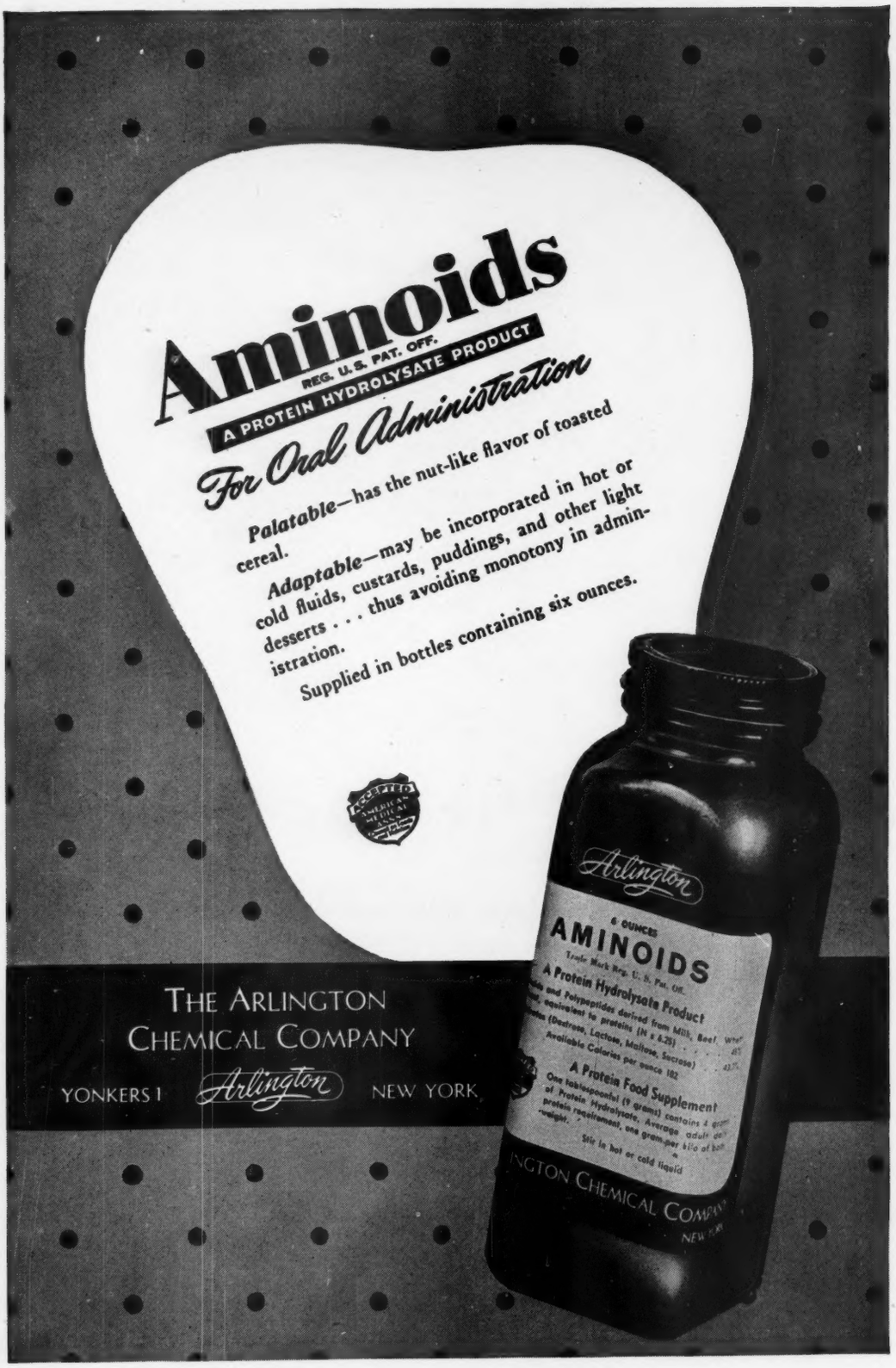
In all cases requiring opiates, Pantopon 'Roche' is preferred by thousands of physicians, for Pantopon contains all the alkaloids of opium in water-soluble form suitable for injection. It is a highly purified preparation freed from the gums, resins and waxes occurring in the crude drug. Pantopon 'Roche' is available in ampuls, in hypodermic and oral tablets, and in powder form.

HOFFMANN-LA ROCHE, INC., ROCHE PARK, NUTLEY 10, N. J.

PANTOPON 'ROCHE'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REG. U. S. PAT. OFF.
A PROTEIN HYDROLYSATE PRODUCT
For Oral Administration
Palatable—has the nut-like flavor of toasted cereal.
Adaptable—may be incorporated in hot or cold fluids, custards, puddings, and other light desserts . . . thus avoiding monotony in administration.
Supplied in bottles containing six ounces.
THE ARLINGTON CHEMICAL COMPANY
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Arlington
6 OUNCES
AMINOIDS
A Protein Hydrolysate Product
A Protein Food Supplement

BIOLOGICALS, BIOCHEMICALS, PHARMACEUTICALS FOR THE MEDICAL PROFESSION

NATIONAL
DRUG COMPANY

Maintaining Positive Nitrogen Balance

In severe protein depletion, maintenance of a positive nitrogen balance is essential. Yet to achieve this solely by diet is frequently impracticable, if not impossible.

The logical alternative is to supply amino acids—those chief building stones of the protein molecule—in the form of an oral protein hydrolysate—AMINONAT.

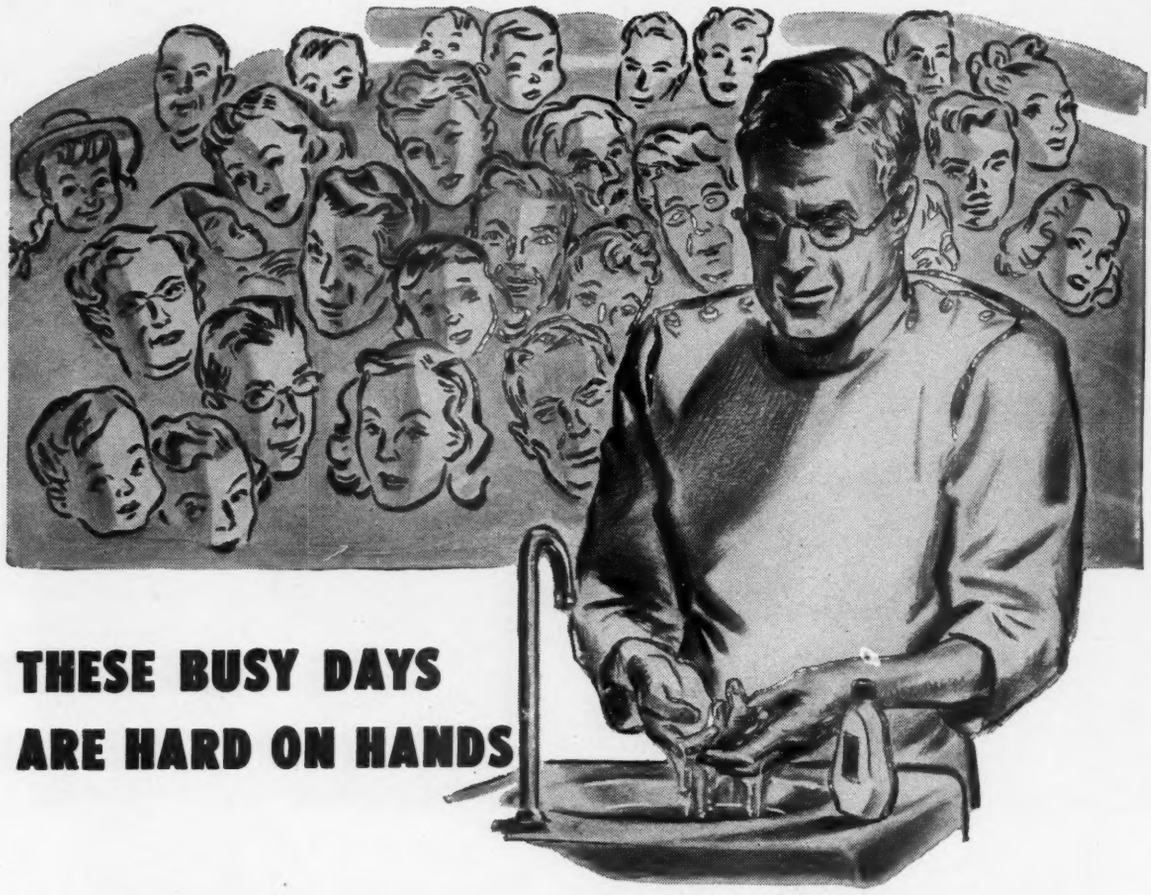
AMINONAT with its high amino acid content lightens the load on the gastro-intestinal tract, thereby making available increased protein without producing gastric distress or causing inconvenience to the patient.

AMINONAT is the preparation of choice whenever oral amino acids are indicated—in the treatment of hypoproteinemia, anemia, allergic states, wound healing, kidney disease, disorders of the gastro-intestinal tract and hypermetabolic states. Write for professional literature to **The National Drug Company, Philadelphia 44, Pennsylvania.**

AMINONAT *contains:* Protein Hydrolysate, 94% (of which the total protein is 70% and the amino acids 40%), sodium chloride, 2% and flavoring agents, 4%. Available in 4 oz., 8 oz. and 1 lb. packages.

Aminonat

THE NATIONAL DRUG COMPANY, PHILADELPHIA, PA.



THESE BUSY DAYS ARE HARD ON HANDS

Are YOURS *receiving proper care Doctor?*

Frequent cleansing with soap and water is often hard on skilled and sensitive hands. Also preoperative "scrubbing up" with hot running water, green soap and alcohol rob the skin of its natural lubricant.

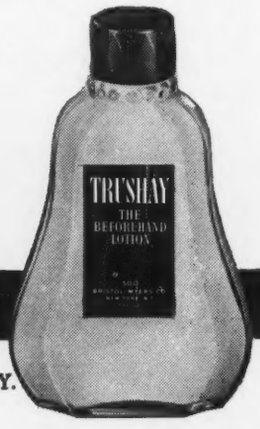
TRUSHAY is widely used by professional men and women as an aid in replacing natural oils and to help keep dermal tissue normal and unbroken.

The principal ingredient of TRUSHAY is lanolin, yet TRUSHAY is neither sticky nor greasy, and is delightfully fragrant.

Smoothed on just before washing, TRUSHAY is just what is needed to help protect skilled, sensitive hands and keep them soft, smooth and supple. TRUSHAY is well adapted to the needs of the physician, the nurse and the technician.

TRUSHAY

THE "BEFOREHAND" LOTION



A Product of BRISTOL-MYERS COMPANY, 19 NF West 50th Street, New York 20, N. Y.

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in

★ BURNS ★ WOUNDS

★ ULCERS ★ SKIN GRAFTS

Clinical evidence continues to accumulate indicating the value of topically applied vitamins A and D in the treatment of lesions involving tissue destruction.

In a series of 713 cases so treated, including burns, denudations, avulsive injuries, osteomyelitis, sloughing and infected abdominal wounds, chronic ulcerations, abscesses, donor and recipient skin grafting sites, and others, Daughtry* reports:

MORE RAPID HEALING—formation of granulation tissue—epithelization.

LOW INCIDENCE of infection—scarring—contractures.

LESS PAIN in certain burn cases—less sedation required; less skin grafting.

*Daughtry, DeWitt C.: Cod Liver Oil Ointment in Surgery, Surgery, *18*:510-515 (Oct.) 1945.

White's
VITAMIN A and D OINTMENT

was used in the treatment of many of these cases. The author comments: "It proved of particular value when applied to donor and recipient skin grafting sites."*

White's Vitamin A and D Ointment provides vitamins A and D derived from fish liver oils, present in ratio as found in cod liver oil, in an appropriate lanolin-petrolatum base; free from unpleasant odor and excessive oiliness. Keeps indefinitely at ordinary temperature.

½ oz. tubes, 8 oz. and 16 oz. jars and 5 lb. containers.

Ethically promoted—not advertised to the laity. White Laboratories, Inc., Pharmaceutical Manufacturers, Newark 7, N. J.

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The New, Completely Rewritten, 3rd Edition

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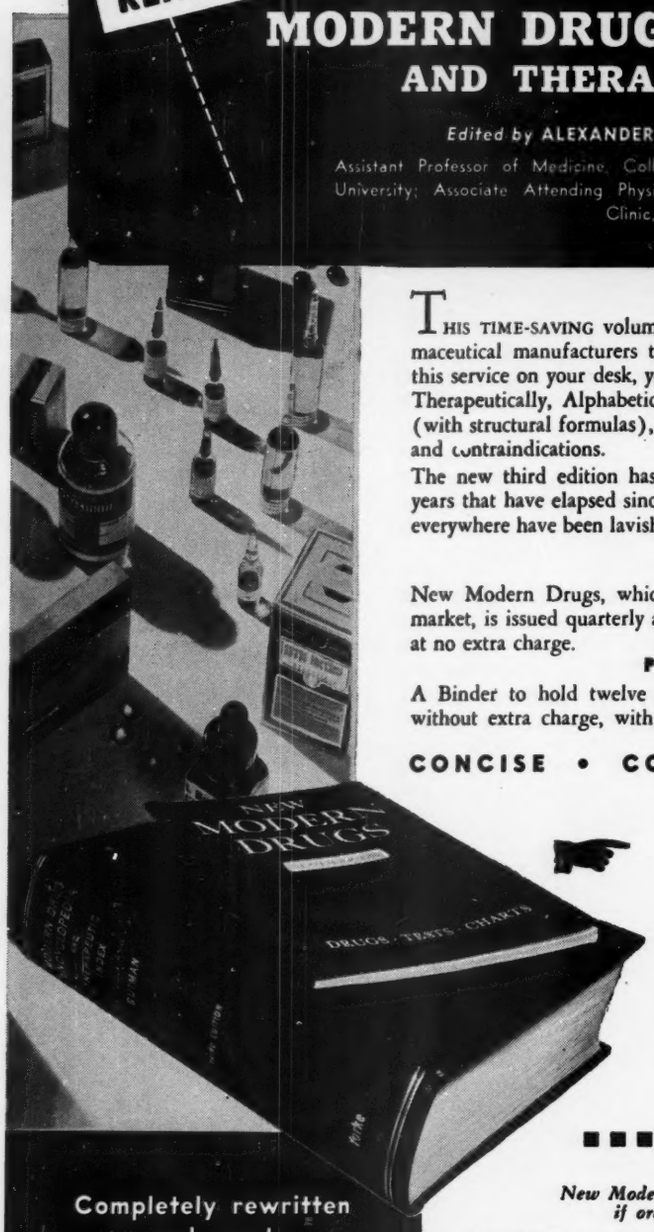
Assistant Professor of Medicine, College of Physicians and Surgeons, Columbia University; Associate Attending Physician, Presbyterian Hospital and Vanderbilt Clinic, New York



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IN THE CORRECTION OF HYPOCHROMIC ANEMIA

More than Iron Alone MAY BE NEEDED

Hypochromic anemia, whether caused by inadequate food intake, increased nutritional requirement, or by blood loss, usually tends to perpetuate itself. Anemia engenders anorexia, hypochlorhydria and impaired digestion. This interferes with adequate intake, absorption and utilization of iron and other essential nutrients, thus intensifying the anemic state.

For the speedy correction of the anemia syndrome and its associated multiple nutritional deficiencies, iron alone is usually inadequate. *All* the lacking essential nutrients must be supplied, by both diet and appropriate medication.

★ **Heptuna**

a **ROERIG** *Preparation*

Heptuna presents a convenient and effective means of treating hypochromic anemia by supplying not only iron, but also other metabolic essentials frequently needed.

EACH CAPSULE CONTAINS:

★ **The Most Readily Available Form of Iron**

Ferrous Sulfate U.S.P. ..... 4 ½ Grains

★ **Natural Fat-Soluble Vitamins**

Vitamin A (Fish-Liver Oil) ..... 5,000 U.S.P. Units
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★ **B-Complex Vitamins**

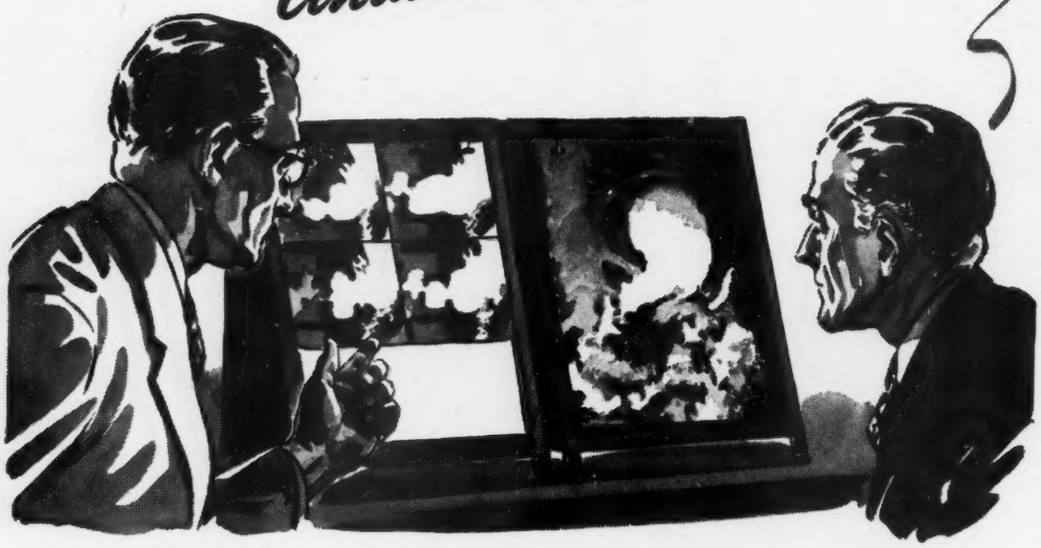
Vitamin B_1 (Thiamine Hydrochloride) ..... 2 mg.
Vitamin B_2 (Riboflavin) ..... 2 mg.
Vitamin B_6 (Pyridoxine Hydrochloride) ... 0.1 mg.
Calcium Pantothenate ..... 0.333 mg.
Niacinamide ..... 10 mg.

★ **Whole Natural B Complex**

Derived from Liver Concentrate (Vitamin Fraction) and Dried Yeast U.S.P.

J. B. ROERIG & COMPANY • 536 Lake Shore Drive • Chicago 11, Ill.

A Non-Systemic Antacid and Protective



IN peptic ulcer, Kamadrox affords rapid relief of epigastric discomfort and prompt objective improvement. It promotes healing of the ulcer crater because of the specific influence of its three ingredients (*Magnesium Trisilicate,* 50%; *Aluminum Hydroxide,* 25%; *Colloidal Kaolin,* 25%).

Kamadrox exerts prompt, profound, and sustained acid-combining power. Yet it is systemically inert so that it cannot lead to alkalosis or secondary acid-rise. By forming a coating over the ulcer crater, it provides mechanical as well as chemical protection. Kamadrox is astringent, demulcent, and adsorbent. Its ease of administration and pleasant taste are appreciated by the patient and assure his cooperation in carrying out the ulcer regimen.

KAMADROX

KAMADROX POWDER is supplied in 4-oz. and 1-lb. jars.

KAMADROX TABLETS are supplied in bottles of 100, 500, 1,000 and 5,000. Each tablet contains: *Magnesium Trisilicate,* 4 gr.; *Aluminum Hydroxide,* 2 gr.; *Colloidal Kaolin,* 2 gr.

KAMADROX LIQUID. Each fluidounce represents 64 gr. (4.15 Gm.) magnesium trisilicate and 32 gr. (2.1 Gm.) kaolin colloidal, suspended in 3½% aluminum hydroxide gel, aromatics, q.s.

THE S. E. MASSENGILL COMPANY

Bristol, Tenn.-Va.

NEW YORK • SAN FRANCISCO • KANSAS CITY

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PROTEIN

*"As a Prophylactic and Therapeutic Agent"**

"There are many opportunities for practical dietary application of the advances in our understanding of protein economy. The provision of liberal protein diets in instances where the value of protein has been demonstrated offers both prophylactic and therapeutic advantages to the patient."

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"Protein is one of the essentials for which there is greater need.... The growth needs of the fetus must be met and if adequate dietary protein is not supplied, the mother's protein stores will be drained.... The demands for protein during lactation are even greater.... One further reason for providing a liberal supply of protein... lies in the favorable effect which protein has upon calcium absorption."

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"... actual improvement in the well-being of nephritic patients may be observed following the use of high-protein diets both in adults and children. In some instances functional capacity of the kidneys has been reported to improve on such a regime.... For the treatment of nephrosis the same dictum holds true, only to a greater extent..."

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"For many years carbohydrate alone has been considered the one substance capable of providing protection for the liver cells. ... Protein has now been shown to be considerably more effective in this respect. ... The effect of protein on the liver is not only reparative, but also definitely protective against the agents known to be toxic to the liver...."

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"... a rather high incidence of moderate hypoproteinemia in peptic ulcer cases has been reported. Any ulcer, be it peptic, infectious, or the result of pressure and circulatory changes, represents a loss of tissue and requires protein for rebuilding...."

OTHER CONDITIONS

"In old age, for instance, all too often the food intake deteriorates to soft foods of limited variety.... Yet in such instances the same protein requirements for maintenance of the numerous body functions are still in effect.... Protein may also be lost because of its excessive breakdown under the influence of fever or heightened body metabolism, and negative nitrogen balances may develop, as for instance in hyperthyroidism....

"With the demonstration of this increased importance of protein dietetic therapy, it can only be recommended that there be an increased use of high-protein diets."

Among the protein foods of man meat ranks high not only because of the percentage of protein contained, but primarily because the protein of meat is of high biologic quality, applicable for every protein need.

---

*Anderson, Geo. K., M.D.: *The Importance of Protein in Diet Therapy,* J. Am. Dietet. A. 21:436 (July-August) 1945.

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AMERICAN MEAT INSTITUTE

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Ortho-Gynol Vaginal Jelly—the most widely prescribed contraceptive preparation—is now available in a *complete set*, with Ortho diaphragm (sizes 55 to 95) and diaphragm introducer. Thus, when pregnancy is contraindicated, the physician may prescribe—*as one unit*—the complete requisites for effective, intelligent contraceptive practice...with full reliance on quality and patient-acceptance. For those who prefer the cream form—*Ortho-Creme* Vaginal Cream is also available in these handsomely packaged sets.

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Ortho-Gynol
VAGINAL JELLY

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The American Journal of Surgery

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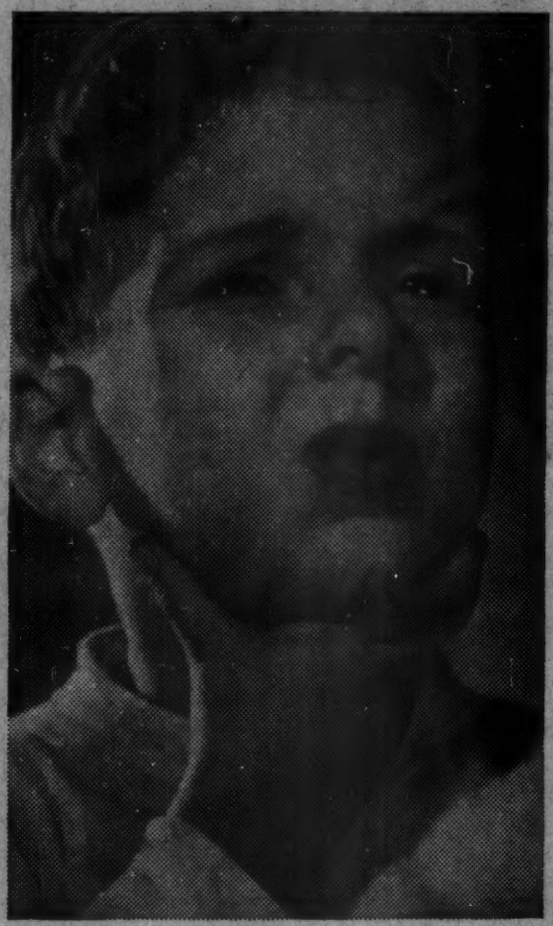
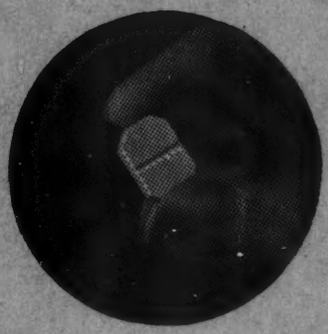
What about Post-Operative care?





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NUPORALS—Available in boxes of 15, and bottles of 100 lozenges.

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